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THE EXTERNAL FRONTOETHMOSPHENOIDAL OPERATION.*†

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We have used the technique to be described in chronic cases only. Most of them had been operated upon one or more times by other methods without relief. Furthermore, we treated them conservatively for a reasonable period of time before deciding to reoperate.

PREOPERATIVE PREPARATIONS.

Local Anesthesia: Three gr. (0.18 gm.) of sodium amytal are given the evening before and another dose in the morning if the operation is scheduled for the afternoon. A hypodermic injection of morphine sulphate gr. ⅙ (0.01 gm.) with scopolamine hydrochloride gr. 1/150 (0.43 mg.) is given one-half hour before the operation.

Avertine: A colonic irrigation and 3 gr. of sodium amytal are given the evening before and 100 mgm. of avertine per kilogram of body weight 15 minutes before the operation. If necessary, this basic anesthesia is supplemented with ether, fed by means of a pressure machine through an ether tube.

About 15 minutes before the operation, the nasal fossa on the side to be operated upon, is packed with pledgets of cotton saturated with a mixture made of equal parts of a 20 per cent

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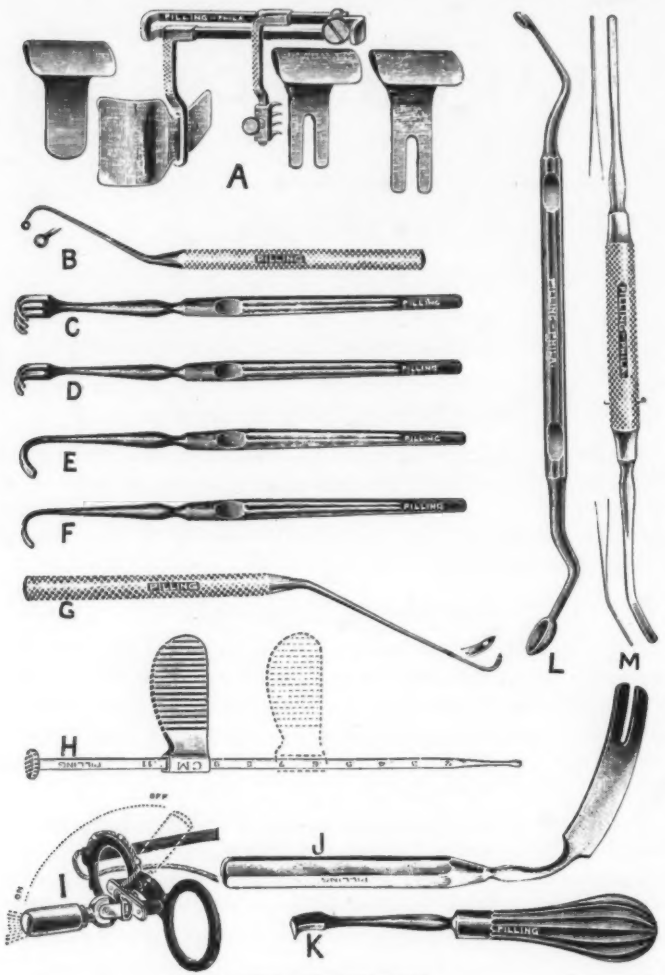
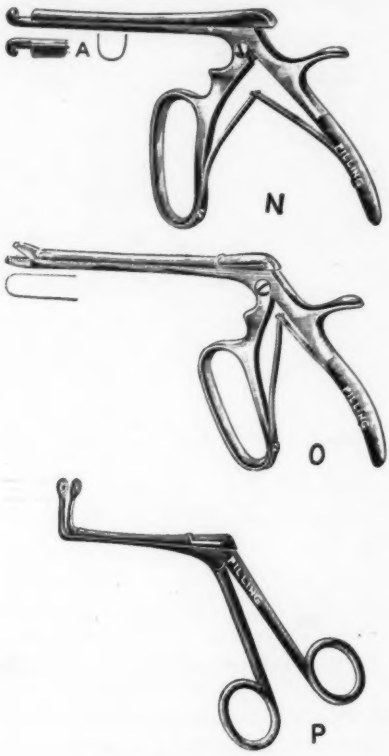


THE INSTRUMENTS.

Fig. 1. (A) Author's self-retaining retractor. (B) Author's ligature-carrying-knot-tier. (C, D, E, F) Author's set of hand retractors. (G) Author's sphenopalatine needle. (H) Author's graduated probe. (I) Light (attached to the spectacles) for illuminating the field of operation. (J) Ferris-Smith slotted hand retractor. (K) Ferris-Smith periosteal elevator. (L) Author's double end retractor. (M) Author's submucous elevator.

solution of cocain hydrochloride and a 1/1000 solution of epinephrine hydrochloride. The packing should be placed as far backward and upward as possible. It is desirable to have it reach the anterior wall of the sphenoid sinus and the superior meatus, if possible, in order to anesthetize the sensory nerves at their points of exit. This packing reduces the intranasal bleeding to a minimum.



THE INSTRUMENTS.

Fig. 2. (N) Ferris-Smith up and downward punch. (O) Ferris-Smith forward biting punch, modified by author. (P) Ferris-Smith sponge forceps.

Instruments: The most suitable instruments are the self-retaining retractor, the ligature-carrying-knot-tier,[1] the slotted hand retractor and the sphenopalatine needle.[2] We use additional instruments which, though not absolutely necessary,

substantially reduce the time of the operation and simplify things (see Figs. 1 and 2).

The patient is placed in the semireclining position and the head is fixed with a suitable head rest.

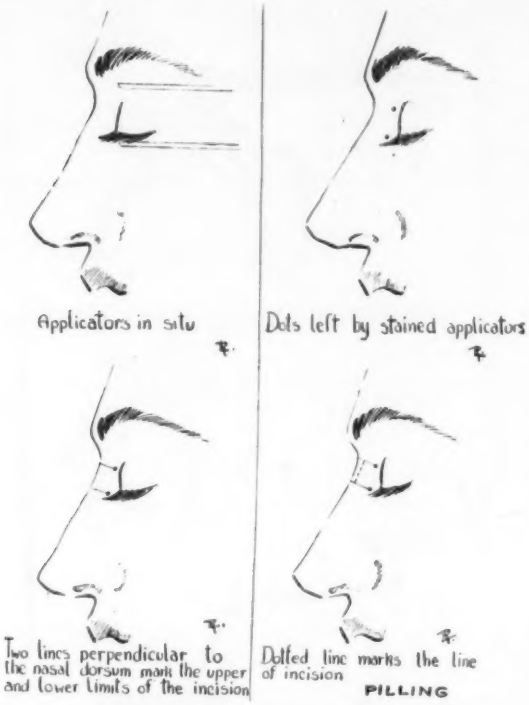

Applicators in situ
Dots left by stained applicators
Two lines perpendicular to the nasal dorsum mark the upper and lower limits of the incision
Dotted line marks the line of incision
PILLING

Fig. 3. The delimitation of the line of incision.

The forehead, lids, cheeks, nose, vestibule, lips and chin are painted with tincture of iodine (3 per cent) which is promptly removed with alcohol.

Delimitation of the Line of Incision: The tips of two wooden applicators are moistened with tincture of iodine. One applicator is placed horizontally against the upper, the other against the lower rim of the orbit, and the stained tips are pushed medially to the side of the nose where two small marks are made.

A line is drawn from each mark perpendicularly to the nasal dorsum. Another line, uniting the two perpendicular ones, is drawn parallel to the nasal dorsum. This indicates the line for the incision (see Fig. 3). We use such a high skin incision to avoid the blood vessels which may cause some annoying bleeding and to obtain a fair sized periosteal flap (see Fig. 5).

Local Anesthesia: A 2 per cent solution of novocain containing 25 mn. (1.5 cc.) of a 1/1000 solution of epinephrine hydrochloride to the ounce (30 cc.) is used. The needle is inserted at the lower end of the line of incision (see Fig. 4A) along which it is slowly advanced upwards until it reaches the roof of the

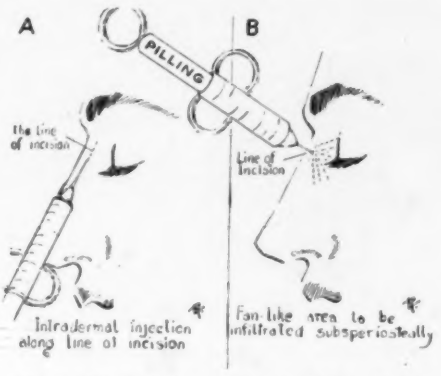


Fig. 4. The technique for the local injection.

orbit. The solution is injected gradually intradermally at first, then subperiosteally. The needle is withdrawn and reinserted in the middle of the line of incision until it reaches underneath the periosteum. It is then advanced perpendicularly, along the inner wall of the orbit, for 3 cm. Next the needle is partly withdrawn and, through the same puncture hole in the skin, the orbitonasal wall is infiltrated along a fan-like area between the upper and lower orbital rims (see Fig. 4B). The needle may impinge against small bony ridges, from which it should be gently disengaged. About 3 ccm. of solution are sufficient. The infiltrated area is gently massaged for a few minutes.

The Incision: The knife is held at a right angle to the side of the nose and the incision is made through the skin. The

underlying periosteum is conserved to be used later as a flap to support the orbital contents and maintain the lumen of the nasofrontal duct. The skin,[3] the mucosal[4] and the osteoplastic[5] flaps recommended by others have not been successful in our hands.

After the skin has been dissected posteriorly for about 1.5 cm., the anterior margin of the incision is retracted toward the nasal dorsum and a second incision is made through the periosteum parallel to the primary skin incision, 2 mm. anterior to it.

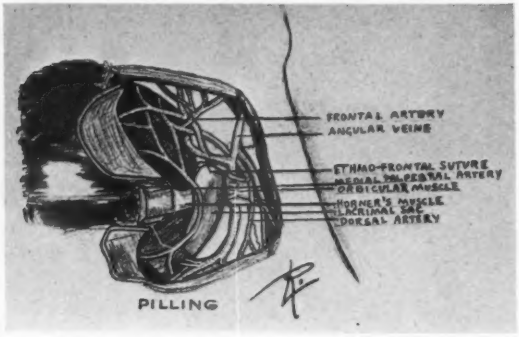


Fig. 5. The relationship of the superficial blood vessels and the lacrimal sac to the incision.

The periosteal elevation is started at the upper third of the incision (see Fig. 6A). At this level the underlying bony surface is smooth and the periosteum but loosely attached; whereas, at the lower two-thirds the bone is rough, the periosteum firmly attached and it dips in along the suture lines. The elevation is made with downward sweeping movements, followed up by forward ones (see Fig. 6B). It is well not to go into the orbit beyond 3 cm. for the reason that the anterior ethmoidal artery is usually found within this distance.

The most important anatomical landmark of this region is the suture-line between the lower edge of the frontal bone and the upper edges of the ascending process of the maxillary bone, the lacrimal bone and the os planum. This line begins

at about 1 cm. above the inner canthus of the eye and continues more or less directly backward. The foramina for the anterior and posterior ethmoidal arteries open in the lower edge of the frontal bone, almost within this suture line. The upper limit of the lacrimal fossa and the dome of the lacrimal sac are at the same level. The optic foramen, located in the sphenoid bone, is 1 cm. directly behind the posterior end of this line. Therefore, in separating the periosteum from the orbitonasal wall it is well to keep at least 1 cm. above the inner canthus of the eye. Below this level one may fall into many difficulties, namely: *a.* The resistance of the periosteum, where it dips

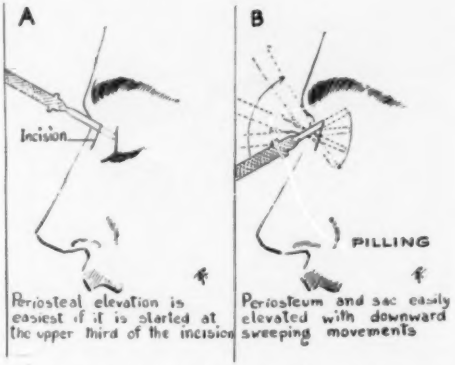

A
Incision
Periosteal elevation is easiest if it is started at the upper third of the incision
B
PILLING
Periosteum and sac easily elevated with downward sweeping movements

Fig. 6. The technique for the periosteal elevation.

down into the suture-line, may result in lacerations with protrusion of orbital fat; *b.* the anterior ethmoidal artery may be injured; *c.* the anterior palpebral ligament, which is attached to the anterior lacrimal crest, will be at such an angle that the periosteal elevator will slip along the outer surface without separating it; *d.* the lacrimal sac may be injured; *e.* the obstructing posterior lacrimal crest, which in most of the cases stands out forming an acute angle with the bottom of the fossa, will interfere with the advance of the periosteal separator; *f.* Horner's muscle (palpebral ligament) which is attached to the posterior lacrimal crest will be at such an angle that one can not easily separate it. On the other hand, if one begins the periosteal elevation above the level of the suture-line and gradually advances the separator downward and forward, the periosteum will easily be elevated; the anterior

ethmoidal artery will be avoided; the posterior lacrimal crest, whose orbital face is a smooth continuation of the inner orbital wall, will not offer any resistance; Horner's muscle will be engaged by the separator at such an angle that it will be easily separated from its attachment to the posterior lacrimal crest. The lacrimal sac will be easily lifted out of the fossa without any danger of being injured; the anterior palpebral ligament will yield to slight pressure of the separator and finally a few gentle downward sweeping movements, each followed by a forward one, will easily expose the upper third of the inner orbital wall.

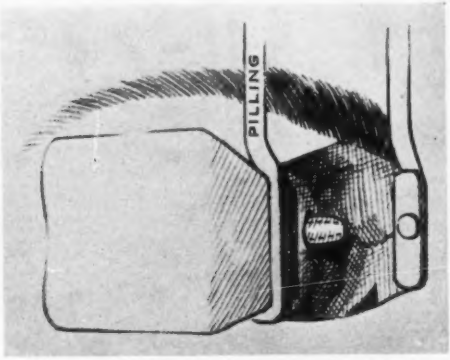


Fig. 7. The sheath containing the anterior ethmoidal artery and nerve.

During this elevation it is advisable not to use much force; rough manipulations may tear the periosteum or break through the thinnest part of the orbitonasal wall (os planum) thus rendering the dissection difficult.

After the elevation of the periosteum has progressed to the depth of 3 cm., a slotted hand retractor is introduced and search is made for the anterior ethmoidal artery. In this region the periosteum is so loosely attached, that the pressure of the hand retractor is sufficient to separate it from the bone. As the periosteum peels off beyond the advancing tip of the retractor, a pearly sheath, which contains the anterior ethmoidal artery and nerve, can be seen crossing diagonally backwards from the eye to the orbitonasal wall (see Fig. 7). It is gently worked into the seat. When it is well engaged, a

slightly firmer retraction exposes a greater length of sheath. The periosteum is elevated behind the sheath before the suture-carrier is passed around it; frequently a flat web of adherent periosteum surrounds the sheath at the foramen.

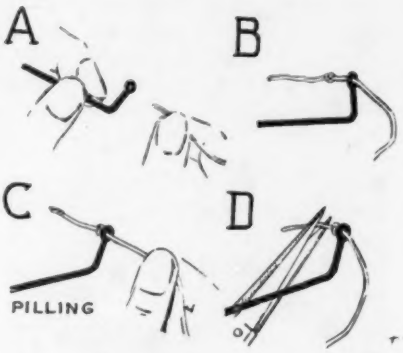


Fig. 8. The threading of the suture-carrying-knot-tier.

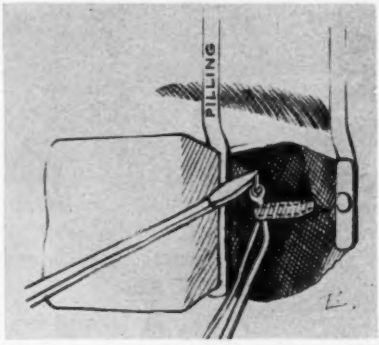


Fig. 9. The suture-carrying-knot-tier has been passed around the sheath and a forceps is about to grasp the free end of the catgut.

Ligation of the Anterior Ethmoidal Artery: The suture-carrying knot-tier is threaded with No. 0 catgut (see Fig. 8A), which should be first soaked in warm water to render it pliable. A knot is placed at about 1 cm. from that end of the catgut which points towards the shank of the instrument (see

Fig. 8B). By pulling on the long end, the knot will become anchored at the eye of the instrument (see Fig. 8C). The end, nearer the knot, is cut to 1 cm. length (see Fig. 8D) and placed in that position which makes it easiest to grab with the forceps. The sheath, containing the artery and nerve, is held on the stretch by the retractor, while the ligature-carrier is passed around it from below. The short end of the catgut as it appears behind and above the sheath, is grasped with a Hartmann alligator forceps (see Fig. 9). While the tier-carrier is held in place, to protect the artery from the saw-like action of the

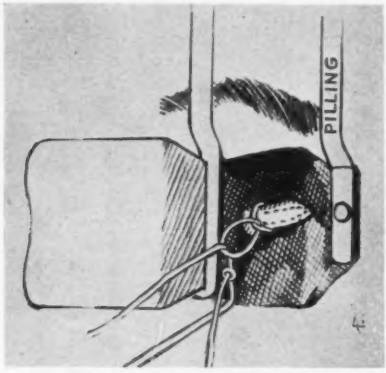


Fig. 10. The knots are slid into position and made tight with the tier-carrier.

catgut, the knotted end is pulled out well beyond the external opening of the wound in order to avoid the necessity of unthreading the instrument within the orbit. The stiffness of the catgut and its tendency to stick within the eye of the carrier renders unthreading difficult.

Outside of the orbit, a knot is formed between the tier-carrier and the sheath, then it is pushed in position and tightened with the same instrument. The ligature is placed about midway between the orbital contents and the orbitonasal wall (see Fig. 10). The sheath is severed between the ligature, and the orbitonasal wall.

Next, the periosteal elevation is continued beyond the artery and along the roof of the orbit where the tendon of the superior oblique muscle is separated from its pulley. One

should bring into view the lower end of the lacrimal fossa and the suture-line between the anterior margin of the ascending process of the superior maxillary bone and the posterior margin of the nasal bone.

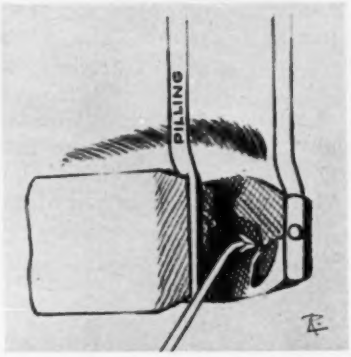


Fig. 11. The first opening into the orbitonasal wall is made beyond the posterior lacrimal crest.

Fig. 12. With a heavy punch the bone is bitten away along a line perpendicular to the nasal dorsum.

The nasal packing is now removed and the bone work is started.

Beyond the posterior lacrimal crest, an opening is made through the lamina papyracea (see Fig. 11). With a Ferracci

this opening is made large enough to admit a stout upward biting punch. The removal of the inner orbital wall is carried forward, along a line perpendicular to the nasal dorsum. The lacrimal bone and the ascending process of the maxillary bone are removed far enough, in some cases, to include a part of the nasal bone itself (see Fig. 12). From the upper extremity of the opening just made the removal of bone is continued in the upward direction along a line parallel to the nasal dorsum (see Fig. 13). At the upper end of this opening the frontal sinus is found. A window, 1.5 cm. in diameter, is placed into the anterior wall of the sinus and the floor is completely removed (see Fig. 14). Where the sinus is large, the original incision is extended upward and curved along the rim of the

Fig. 13. At about 0.5 cm. from the posterior margin of the nasal bone the biting is continued upwards along a line parallel to the nasal dorsum.

orbit. The lining mucoperiosteum of the frontal sinus is thoroughly soaked with a solution of 5 per cent picric acid in equal parts of acetone, alcohol and distilled water, then rubbed off with dry pledgets of gauze or applicators. A small laryngeal mirror is used to inspect the inner surface of the anterior wall.

The lamina papyracea is completely removed, beginning at the original opening and advancing posteriorly (see Fig. 15). Usually it is not necessary to go beyond the posterior ethmoidal artery, but, if necessary, one may reach as far posteriorly as the common tendon of the extraocular muscles. In this event, the posterior ethmoidal artery is also ligated.[6]

Fig. 14. A window is made in the anterior wall of the frontal sinus.

Fig. 15. The removal of the lamina papyracea is carried from the initial anterior opening backwards.

The lower third of the orbitonasal wall is left *in situ* with smooth edges (see Fig. 16). The anterior ethmoidal cells are removed with a special curette along the lower third of the ascending process of the maxillary bone. The curette is intro-

duced either through the nostril or through the external opening (this is the only place where a curette is used).

Most of the probing is done with the tier-carrier which is safe on account of its blunt end. This instrument is also used through the nostril for pushing the nasal mucosa, polypi, or middle turbinate remnants through the orbital opening where they are grasped and cut with an ethmoid punch (see Fig. 17). It is well not to pull on the mucosa at any time.

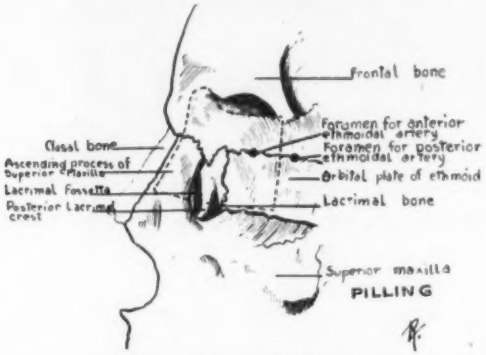


Fig. 16. The dotted line delimits the bony area removed.

The use of the carrier to push the intranasal structures out through the orbital opening, gives one a feeling of security as long as the cutting is done outside of the nasal cavity (in the orbit).

The location of the anterior wall of the sphenoid is determined after the following manner: The tier-carrier is introduced along the floor of the nose to the posterior pharyngeal wall. It is then brought upward to the roof of the nasopharynx (see Fig. 18A), then forward around the arch of the choana, finally upward along the anterior wall of the sphenoid (see Fig. 18B). To remove the obstructing posterior end of the middle turbinate, or other tissues that may be found lying in front of the sphenoid sinus, the tier-carrier is introduced as just described and placed onto the anterior sphenoidal wall, where it is held as a guide while a punch is used through the orbital opening (see Fig. 19). One hand holds the carrier while the other hand operates the punch. In this way the

Fig. 17. Intranasal tissues are pushed out through the external opening, where they are bitten off.

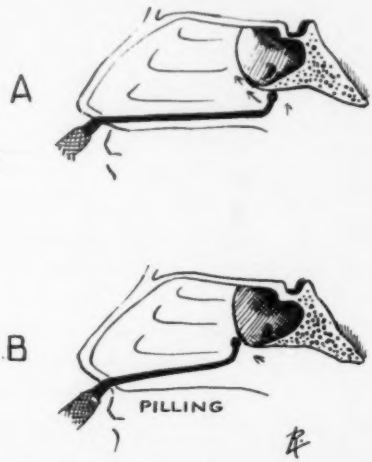


Fig. 18. The technique for the localization of the anterior wall of the sphenoid sinus.

proximity or contact of the two instruments will be easily perceived, thereby affording one a sense of security. This maneuver will protect the dangerous angle formed by the foveolae ethmoidales and the anterior wall of the sphenoid.

It is advisable to have the frontal and the sphenoid sinuses well in evidence before attempting to clean the foveolae ethmoidales, which are in a dangerous region. The foveolae are situated in the frontal bone behind the frontal sinus, extending backward a variable distance, in some cases quite to the sphenoid bone. The external inferior limit of the foveolae is the upper edge of the paper plate or planus, the superior median limit is the outer edge of the cribriform plate (see Fig. 20).

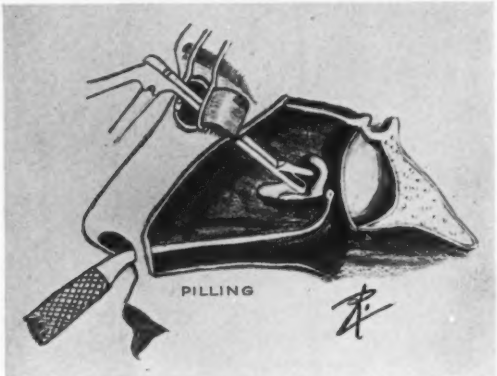


Fig. 19. Structures lying in front of the sphenoid are bitten off while the ligature-carrying-knot-tier is held up against the anterior wall of the sphenoid.

In this area the dura may be easily exposed, therefore one must be very careful in probing. Bony ridges should be levelled off only after their outline is clearly seen.

The mucosa lining the cells is first saturated with the picric-acetone solution, then gently rubbed off with dry pledgets of gauze or cotton wound applicators. To obtain the best results, the foveolae should be cleaned to the bone. The cleaning is started from the posterior wall of the frontal sinus, gradually advancing downward and backward, along the foveolae ethmoidales, as far as the sphenoid sinus.

The cribriform region is more dangerous than the foveolae. The outer limit of the cribriform plate is marked by a thin, irregular ridge (the cut-off attachment of middle turbinate); the inner limit is marked by the nasal septum.

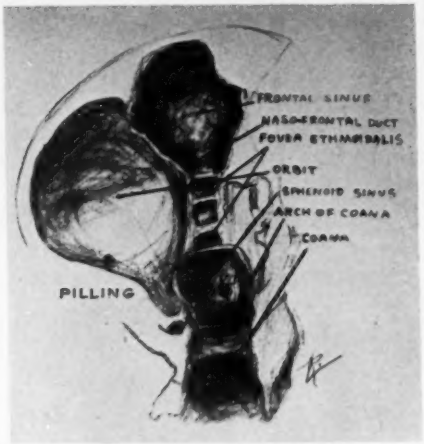


Fig. 20. Cross-section below the cribriform plate.

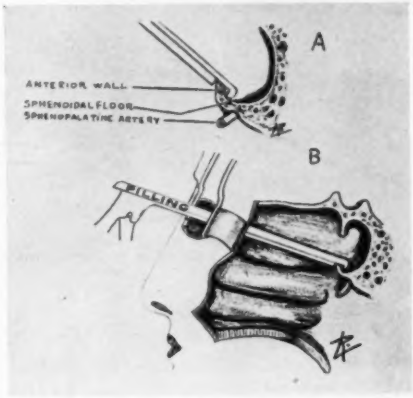


Fig. 21. The floor of the sphenoid sinus stops the punch at a safe distance from the sphenopalatine artery, and the instrument works at such an angle that the resulting cut will be on a protective slant.

The picric acetone solution is effective in cleaning small corners. We have applied it directly to the dura without harm.

If the dura should be accidentally exposed, the opening is made larger as it is done during a mastoidectomy.

After the anterior sphenoidal wall has been well exposed, the ostium is made large enough to admit a down-biting punch. This is introduced through the orbital opening; through this approach the instrument will meet the anterior sphenoidal wall at the most favorable angle for its removal (see Fig. 21A). The sphenopalatine artery is contained within the layers of the mucoperiosteum which covers the undersurface of the sphenoid sinus. It runs transversely below the level of the floor of the sinus. A small bony groove marks the course

Fig. 22. Taking a bite off of the outer corner of the floor of the sphenoid while the elevator is held between the floor and the mucoperiosteum to protect the sphenopalatine artery.

of the artery. Thus, the anterior wall of the sphenoid can be safely bitten off down to the floor. The down-biting punch is thus prevented from doing any damage to the artery (see Fig. 21B).

The lining mucosa of the sphenoid sinus is saturated with the picric acetone solution and gently rubbed off with dry cotton wound applicators or small pledgets of gauze. No currettage is done within the sinus cavity.

The floor of the sphenoid sinus is not removed in every case. Generally we are satisfied to remove the anterior wall

Fig. 23. The sphenopalatine needle ready to pierce the mucoperiosteum.

Fig. 24. The mucoperiosteum has been pierced and the free end of the catgut extends beyond the floor, where it is easily seen and grasped with a forceps.

alone. Should it regenerate, it can be subsequently bitten off at the office. The floor is removed when it slopes obliquely backward, especially in those cases where the patient's vision is in danger.

Tying of the Sphenopalatine Artery: The mucoperiosteum covering the under (nasopharyngeal) surface of the sphenoidal

floor is gently elevated with a thin bent submucous elevator.[7] After the elevation is completed, the elevator is held in place at the outer third of the sphenoidal floor, to protect the mucoperiosteum containing the artery and to guide the punch with which a bite is taken off of the floor (see Fig. 22). The sphenopalatine needle[8] is threaded like the tier-carrier and it is introduced through the nostril. The needle will easily pierce the mucosa stretched across the punched out nick of the floor (see Fig. 23). The knotted end of the catgut will emerge beyond the floor in such a position that it can be easily seen

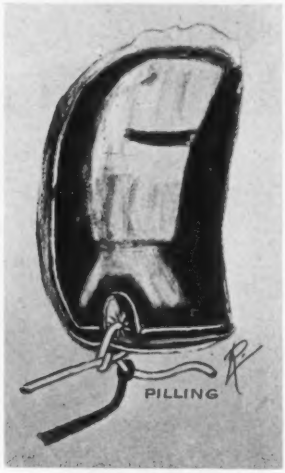


Fig. 25. Tying the sphenopalatine artery with the tier-carrier.

and grasped with a Hartmann alligator forceps which is introduced through the external opening (see Fig. 24). After the needle has been withdrawn, one end of the catgut will be coming out of the external opening while the other end will be lying on the floor of the nose; this end is brought out through the external opening. The tying is done with the tier-carrier (see Fig. 25). The Shuster tonsil needle could also be used for this ligature. After the sphenopalatine artery has been tied, the floor and the underlying mucosa are both bitten off between the ligature and the septum (see Fig. 26).

We consider the operation complete when: *a.* The floor and the lining mucosa of the frontal sinus have been completely removed; *b.* the ethmoidal cells have been all exenterated; *c.* the posterior wall of the frontal sinus, the foveolae ethmoidales and the roof of the sphenoid have been reduced to a continuous white bony area; *d.* the mucosa has been completely removed from the sphenoid sinus.

Fig. 26 The floor and the underlying mucoperiosteum are both bitten away between the septum and the ligature.

At the end the field of operation is illuminated with a light introduced through the nostril and the assistants are asked to inspect the field from every angle for any vestige of mucosa or pathological tissues that may have escaped the attention of the operator. The largest rubber tube that can be introduced through the nostril is used for drainage. Three to four strands of doubled up silkworm gut are placed within the lumen of the tube (see Fig. 27A). The free ends of the strands of gut emerge out of the lower opening of the tube, while the doubled up ends emerge out of the upper opening which is placed into the frontal sinus. The moisture makes the curved ends of the gut spread like a basket (see Fig. 27B). The advantage of this loaded drainage tube is that the strands of silkworm gut will prevent any clogging and will give constant drainage by capillary action. If necessary, the strands may be easily replaced while the tube remains *in situ.*

After the drainage tube is firmly engaged into the frontal sinus, the retractor is withdrawn and the orbital contents is allowed to drop back into place. The anterior margin of the periosteal flap is pulled beyond the posterior margin of the nasal bone where it is sutured with catgut to the posterior periosteal margin (see Fig. 28A and B). Interrupted sutures with hair or dermal are used for the skin; three are usually sufficient. The eye is covered with fluffy pads which are held in place by a tight bandage.

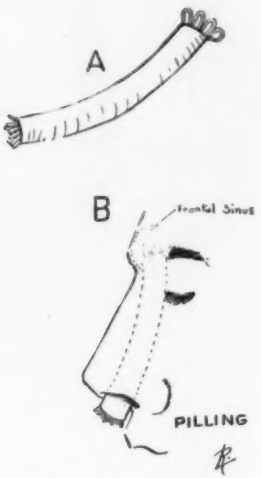


Fig. 27. (A) The drainage tube loaded with silkworm gut. (B) The tube in place.

Operative Accidents: In three cases the dura has been accidentally exposed in the region of the foveolae ethmoidales. The exposed area was made wider and the patients made an uneventful recovery.

Occasionally the orbital periosteum has been lacerated and the orbital fat has protruded into the nasal cavity without any ill effects. Of course it is desirable to avoid this accident which renders the operation quite messy, and exposes the orbital contents to the infections existing in the sinuses. The protruding orbital fat also interferes with the view.

In a few instances, the anterior ethmoidal artery and the sphenopalatine artery have been lacerated or cut, before ligation, without any severe bleeding.

After Care: The patient is made comfortable with sedatives. The bandage is removed in 24 hours. The stitches are covered with collodion; iced saline pads and boric acid douches are applied to the eye immediately after the bandage has been removed. The patients are usually allowed to sit up on the third day, and discharged from the hospital on the seventh day.

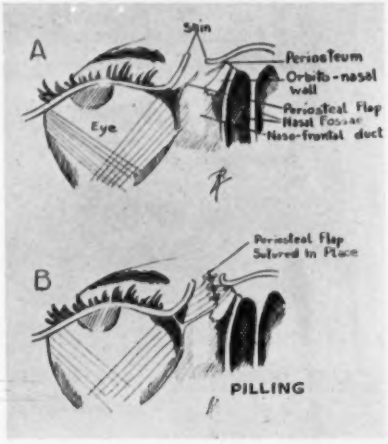


Fig. 28. (A) The periosteal flap elevated. (B) The periosteal flap sutured in place.

COMMENT.

There is swelling and pain over the frontal region and about the eye which lasts from three to six days. A few cases have had diplopia lasting for five to seven days.

Copious, thick, tenacious nasal discharge occurred after the removal of the drainage tube. Later large crusts formed for a period of from two to four months after the operation. The crusts gradually diminished and then disappeared, while the discharge, especially the postnasal, lasted longer.

Comparatively few cases have had some granulations along the nasofrontal duct; applications of strong silver nitrate solutions readily reduced them. The nasofrontal duct has

remained opened in some of the cases and flattened or occluded in others. In the cases with narrowed nasofrontal ducts the drainage has been poor and the headaches have been severe and persistent.

In the cases where the floor of the sphenoid sinus was not removed, the anterior wall had a tendency to reform.

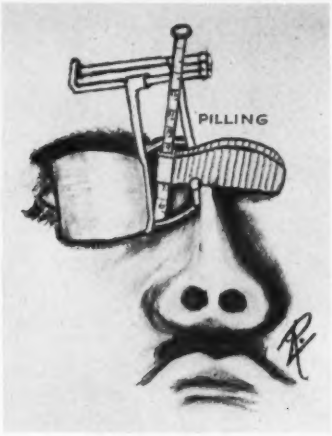


Fig. 29. Measuring the depths of the different structures from the nasal dorsum, with the graduated nasal probe.

A great empty area in the middle and superior meati results from this operation, but none of the cases have complained of a dry throat. We believe that the inferior turbinate, the septal mucosa, and the orbital periosteum overhanging the remaining lower third of the naso-orbital wall, humidify and divide the air currents in such a way that dryness of the throat is prevented.

Lacrimation occurred in a few cases, but it subsided spontaneously.

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1713 Pine Street.

THE TREATMENT OF CHRONIC SINUS INFECTION WITH UNDENATURED BACTERIAL ANTIGENS.

DR. F. C. KRACAW, Oakland.

The use of vaccines as an adjunct to the treatment of chronic sinusitis has been a debatable point for many years past. It is a common experience among specialists in nose and throat work to find the results of vaccine administration extremely erratic and as a rule one sees just enough of good accomplished to maintain interest in this sort of therapy without adopting it for routine use. Some time ago Krueger conceived the idea that a large portion of the failures accruing to vaccine treatment depended upon alterations undergone by the bacterial antigenic complexes during preparation of the vaccine. Krueger and Nichols[1] have shown that proteins — the major antigenic substances of the cell — exist in the live bacterium in the native form and that these proteins are rapidly denatured by temperatures over 40° C. Since the usual treatment for killing bacterial suspensions involves exposure to 60° C. for one hour, they point out that orthodox vaccine therapy has been carried out practically altogether with cellular proteins in a form entirely different from that existing in the live bacterium.

In order to avoid denaturation in the preparation of bacterial antigens Krueger[2] developed a technique for physically disrupting the living cells. Essentially the procedure is as follows: *1.* Growth of mass culture on suitable media; *2.* Harvesting the cultures with successive washing in isotonic Locke's solution to remove metabolites; *3.* Grinding in a special ball mill to disrupt the cells; *4.* Filtration through acetic collodion membranes to retain intact cells and permit passage of bacterial fragments and substances in solution; *5.* Standardization of the filtrate on the basis of native protein content.

In a previous communication[3] the present writer reported his experience in treating a group of patients suffering from chronic sinus infections with autogenous undenatured antigens prepared according to the Krueger method. Of the 45 patients

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treated improvement was noted in 95 per cent and marked improvement or cure in 66 per cent. Naturally the permanence of the results obtained constitutes a major criterion in passing judgment upon the efficacy of any new clinical procedure. A year after completion of treatment in the original group the patient's present physical status was determined through questionnaires and personal examinations. Of the 30 patients classified originally as cured 28 are still in this group, and of the 13 considered as satisfactorily improved, the entire group has maintained this status. In Table I are listed relevant data pertaining to the first series of cases.

The present report concerns the clinical results with a second group of cases. Before considering the detailed results of this group I should like to discuss briefly the indications for surgery as compared with the indications for immunization, together with the technique of immunization with undenatured antigens.

TABLE I.

Improvement in General Health Due to the Treatment		Susceptible to Colds (An outstanding complaint in 36 cases)		Suffer from Headaches (An outstanding complaint in 31 cases)		Postnasal Drainage (An outstanding complaint in 39 cases)	
Yes	No	Yes	No	Yes	No	Yes	No
27	13	6	30	11*	20	14**	25

*Of the 11 patients who answered "Yes," two said their headaches are as severe as formerly, while the remaining nine say that they are less severe.

**The slightest postnasal discharge led to inclusion in the "Yes" category.

Indications for Surgery: Surgery is definitely indicated:

a. Where there is obvious thickening of the antral lining membrane or polyposis; *b.* Where there is present fibrosis of the mucoperiosteum as evidenced by diffuse cloudiness in the X-ray picture with the presence of inspissated pus upon diagnostic puncture; *c.* Where there is any anatomic interference with ventilation or drainage.

Indications for Immunization: Immunization by the present procedure is indicated in: *a.* Allergic rhinitis of probable bacterial origin. Here the mucous membrane of the turbinates is swollen, boggy, pale and a watery secretion is present in the middle and inferior fossae. *b.* Chronic ethmoiditis and

chronic sphenoiditis. *c.* In chronic infection of the frontal sinuses. Here it has been my experience that immunization directed towards correction of concomitant infection in the other paranasal sinuses will usually permit the frontal sinuses to return to normal. Recovery of the other accessory sinuses accelerates recovery of the nasal mucous membrane; this in turn allows adequate drainage from the frontals to occur and facilitates their recovery.

THE METHOD OF IMMUNIZATION.

The undenatured antigens are made from pathogens isolated by cultures taken from the nasal tract. The technical procedures developed by Krueger for preparing these antigens are described in his papers.[1,2,4,5] The antigens are employed for topical immunization and for general immunization by hypodermatic injection.

1. Topical Immunization. Application to the sinuses every fourth day is effected by the Proetz displacement technique. This is adequate for the treatment of the ethmoidal and sphenoidal sinuses and if properly carried out it results in instillation of some of the antigen into the antra. Where antral involvement is of prime consideration direct instillation, either through openings which have already been made or by puncture, is carried out at intervals during the course of treatment. Preliminary shrinking of the nasal mucous membrane facilitates topical administration and should be carried out conscientiously when the ethmoids and sphenoids are involved. Local reaction manifesting itself in slight nasal obstruction and in some cases by mild headache customarily following the first few instillations. As treatment is carried out the drainage increases in amount and becomes purulent — a favorable indication since the antigen apparently acts by stimulating phagocytosis. Finally the drainage becomes mucopurulent and gradually diminishes in quantity.

2. General Immunization. For the production of general immunity the antigen is administered three times a week over an average of 15 weeks. Frequency of administration is then reduced to once a week for six weeks, then once every two weeks for four or five doses and finally once a month for a period of six months to a year. No set dosage scale has been found to be tenable. Treatment is begun with small doses administered intradermally and subcutaneously; at each

treatment a small intradermal dose is given and the subcutaneous dose is gradually increased until subjective or objective improvement is evident. Considerable caution should be exercised in increasing the amount of antigen given, particularly when arthritic symptoms are present. As soon as it is evident that the patient is responding favorably no effort is made to increase the amount of antigen beyond this level but repeated doses of the same amount are given. The usual initial dose is 0.05 cc. and this can be increased rather rapidly as a rule, to 0.4 cc. or 0.5 cc.

RESULTS.

The second series of cases comprises 62 patients with chronic sinus infection. Table II lists the principal complaints encountered, the sinuses involved, previous surgery and the organisms isolated from the nasal cultures.

The end-results in this group of patients may be classified as follows: *1.* No improvement, 2 cases, 3 per cent; *2.* moderate improvement, 8 cases, 13 per cent; *3.* marked improvement, 16 cases, 26 per cent; *4.* clinical cure, 36 cases, 58 per cent. Total 62 cases, 100 per cent.

TABLE II.

Principal Complaints Encountered	Sinuses Involved	Previous Surgery	Organisms Isolated from Nasal Cultures
Postnasal discharge 54	Ethmoid sinuses 49	Tonsillectomy ..50	Staphylococcus aureus hemolyticus 59
Frequent colds 37	Antra 39	Radical antrum 17 (either one or both sides)	Streptococci 41
Nasal obstruction 24	Sphenoids 22		Hemophilic gram negative rods 13
Headache 20	Frontals 4	Simple antrotomy 3	B. pyocyaneus.. 5
Arthritis 18	All sinuses 8		Pneumococcus.. 3
Eye inflammation and visual disorders 5			
Hay fever and asthma 5			Pfeiffer's bacillus 2
Facial acne..... 2			Friedlander's bacillus 1

It will be seen that 84 per cent of the patients treated showed either clinical cure or marked improvement. This contrasts favorably with the 66 per cent of satisfactory results obtained in the first series of 45 cases.

DISCUSSION.

On the basis of three years'[1] experience with the use of antigens (prepared by Krueger's method) in the topical and general immunization of patients with chronic sinus infections it has become clear to me that this method of treatment constitutes a useful therapeutic modality. It is not a cure-all for sinus infections and cannot replace sinus surgery when obvious indications for surgery exist. Its use requires, first of all, the proper selection of cases and, secondly, patience on the part of both doctor and patient. As a rule a four months' period of active treatment is required and the patient is given occasional treatments over an additional five months' period. In comparing the results of the first series of cases with those obtained in the second series the additional 18 per cent of satisfactory results can be definitely attributed to lengthening the period of immunization.

At first some difficulty was experienced in arriving at what Krueger calls a "maintenance dose," *i.e*, the amount of antigen which serves to produce subjective and objective improvement without any untoward local or general reactions. Experience alone furnishes one with the data upon which to base dosage and it is essential that continuous and careful observation be exercised in ascertaining the effective dose.

It is interesting to note that of 18 patients who presented as the outstanding complaint some type of arthritis, 10 were cured, five were definitely improved, and only three were left unchanged. These results support the theory that a sinus infection can serve as the focus for development of infectious arthritis and they emphasize the necessity for carefully considering the possibility of sinusitis in the clinical examination of patients complaining of arthritis.

Hosmer[6] has recently reported his results using the Krueger antigens in the treatment of chronic sinus infections. Of 50 cases treated he obtained good end-results in 31, fair results in 13, and no improvement in six cases. While these results compare favorably with the two series I have reported they really are particularly significant since all of his patients had been under observation for a number of years and had been subjected to all sorts of treatment before using the antigen. Grouping Hosmer's good and fair results together he reports a total of 88 per cent as compared with 84 per cent in the currently reported series.

SUMMARY AND CONCLUSIONS.

The present paper deals with the treatment of chronic sinusitis by immunization with the undenatured bacterial antigens of Krueger.

A year or more after completion of treatment in a previously reported series of 45 cases, it was found that 28 of the 30 patients considered cured had maintained that status, while the 13 patients classified as presenting satisfactory clinical improvement all remained in this category. A second series comprising 62 patients showed 52 markedly improved or cured, 2 failures, and 8 patients in whom improvement was only moderate.

On a percentage basis the present series revealed 84 per cent patients markedly improved or cured as compared with 66 per cent so classified in the first series. The improvement in results may be ascribed to a modification of technique involving a more persistent immunizing program.

In properly selected cases it is the writer's opinion that topical and general immunization with undenatured bacterial antigens, prepared according to the technique outlined, is the method of choice for the treatment of chronic sinusitis.

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CAVERNOUS HEMANGIOMA OF THE HARD AND SOFT PALATE, ANTERIOR AND POSTERIOR PILLARS OF THE PHARYNX AND THE LARYNX.*

DR. JOHN F. WOODWARD, JR., New York.

Angiomas are classified as vascular tumors, although some pathologists refuse to place them in the tumor class and refer to them rather as malformations.

Angiomas are divided into two groups: *1.* Hemangiomas; *2.* lymphangiomas.

The hemangiomas, in which we are interested, are divided into: *1.* Capillary angioma, angioma simplex, telangectatic angioma, naevi, etc; *2.* cavernous angioma; *3.* angioma simplex hypertrophicum: The capillaries show evidences of proliferation of the endothelial cells and many regard them as true endotheliomas; *4.* to this group should be added pseudo-angiomas, such as bleeding polyps, fibroangiomas, circoid aneurysms, etc.

Cavernous hemangiomas are thin-walled and possess a distinct afferent artery and efferent veins. They do not communicate with the neighboring capillaries. According to Blair, "They occur singly or in groups and show lumps or ridges of distended veins of a dark bluish or greenish black color, which project slightly above the surface and may extend into the subjacent tissue. They are compressible but immediately refill when pressure is released. When the head is held low or pressure is put upon the deep jugular veins or whenever anything else occurs to retard the venous return, they become more distended than usual."

They are usually congenital in origin. Ribbert believes they arise from a rudiment which is destined to form blood vessels.

They are of clinical importance because of their liability to hemorrhage and because they may spread indefinitely, destroying the surrounding tissues.

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*From Dr. E. Ross Faulkner's Throat Clinic, Manhattan Eye, Ear and Throat Hospital, New York.

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In the nasal cavity cavernous angiomas are prone to invade the sinuses, and in the region of the ethmoidal labyrinth are especially dangerous. Angiomas originating on the lateral wall of the nasal cavity are not such a rare condition as textbooks would indicate. They may fill the nose, the sinuses, or the nasopharynx and may erode the bony walls of the nasal cavities causing exophthalmos, nasal obstruction, or even facial disfigurement.

In the larynx the true cavernous angioma must not be confused with the more frequent capillary angioma seen following hematoma due to trauma.

In the tongue they are usually not large, although there have been cases reported in which the whole tongue was involved.

A review of the cases in the Manhattan Eye, Ear and Throat Hospital over the past five years shows only six cases of hemangioma. One involved the orbit. The orbit was explored, but the tumor was not removed. No biopsy was taken and we have only the clinical diagnosis of this case. There was one involving the tonsillar fossa. This case of Dr. D. H. Jones had a biopsy taken and a definite diagnosis made by microscopic examination. The growth was removed by the use of radon implants. There was also one case of capillary hemangioma of the right cord and another of the septum. There have been several angiofibromas, mixofibroangiomas, and angiomatous polyps of the nasopharynx and the larynx. These have been ruled out as not being true hemangiomas, as well as all those cases diagnosed as cavernous hemangiomas but not verified by microscopic examination.

Very little is written in textbooks concerning these tumors of the nasopharynx and larynx. Articles by Heyman and Michulitz, Kummel, Marchief and Oppikofer mention this type of growth in order to emphasize the infrequency of its occurrence. Johnson, in 1865, and Fourine, in 1867, reported vascular tumors of the larynx.

New and Clark, in 1919, at the Mayo Clinic, reported three cases of true cavernous hemangioma of the larynx. Their extensive review of the literature revealed only 52 cases reported.

Crowe and Baylor, in 1923, reported eight cases, six cavernous hemangiomas of the nasopharynx and two angiosarcomas. It is interesting to note that surgery in one of these cases proved quite hazardous, due to excessive bleeding.

Huey, in 1927, reported a recurrent hemangioma of the pharynx and larynx at the New York Academy of Medicine. At that time Dr. D. H. Jones and Dr. F. M. Law discussed the use of iodized oil in the pharynx and larynx for taking X-ray films on these cases. This procedure has since been abandoned due to the inability of the oil to adhere to the walls.

Concerning the treatment, many procedures have been tried. The old method of Wyeth, *viz.*, injecting boiling water, has been abandoned, due to sloughing and possible emboli. If the growth is circumscribed, surgery is the method of choice, although recurrences have occurred following surgical removal. Diathermy, various forms of cautery, and radium have been used. Radium offers the best method of treatment in the majority of cases. Simpson, in 1931, reported the use of radium packs, but knew of no cases where radon implants had been used.

Case Report: Male, white, Italian, age 48 years, admitted Jan. 14, 1935, complaining of hemorrhage from the nose and throat.

Family History: The patient's father died at the age of 72 years from some syphilitic condition. His mother is alive and well. He has four brothers and one sister, alive and well. He is married and has three children. One child died from an accident. There has been no tendency to bleed in this patient or in any member of his family.

Past History: The patient can recall a choking sensation in his throat at the age of 18, 30 years ago. He dates his throat condition from that time, although there was no hemorrhage and the hoarseness, etc., cleared up in a month or so. About 12 years ago, he began having a purulent discharge from his nose and has had a pansinusitis ever since. He has had occasional attacks of hoarseness, difficulty in swallowing, etc., but no hemorrhage until two years ago, when the attacks were associated with varying degrees of hemorrhage. There has been no severe pain and no actual difficulty in breathing except that caused by the hemorrhage. He has had the usual childhood diseases with no unusual complications.

Present Illness: For the past two years the patient has had intermittent hemorrhages from the nose and throat. These hemorrhages have occurred at intervals of one to two months, usually clearing up without medical attention. In the last six months they have become progressively more severe. They are usually brought on by exertion, bending over, etc., and have even occurred during sexual intercourse. In fact, any form of physical exertion causing a rush of blood to the head may bring on hemorrhage. These hemorrhages are associated with hoarseness, dysphagia and some, but not marked, pain in the larynx. The associated hoarseness apparently clears up during

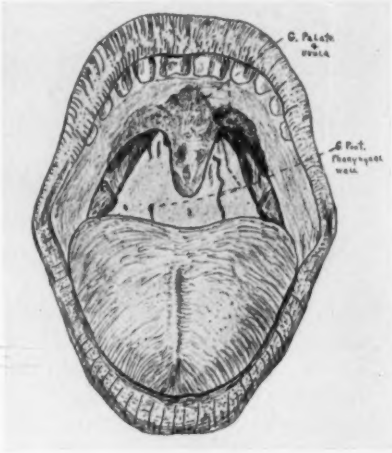

Fig. 1.

the intervals; however, there has been very little improvement in speaking since admission. He has lost 10 pounds in weight during the past year. He is a heavy smoker, about 10 cigars a day, and complains of indigestion after meals and marked constipation (see Fig. 3).

Examination of the nose shows a dry, pale mucous membrane. There is slight deviation of the septum and enlarged and boggy turbinates. Along the anterior and posterior upper portion of the septum and anterior portion of the lower and middle turbinates may be seen small pin point purplish spots. Whether or not these have anything to do with the growth cannot be definitely determined.

After cocainization, nasopharyngoscopic examination of the posterior portions of the turbinates and the nasopharynx reveals beginning polypoid degeneration of the posterior tips and extension of dark dilated vessels from the pharynx. How high up into the vault or sphenoethmoidal recess these extend cannot be definitely determined. The pin point spots seen in anterior examination may be noted here and there (see Fig. 1).

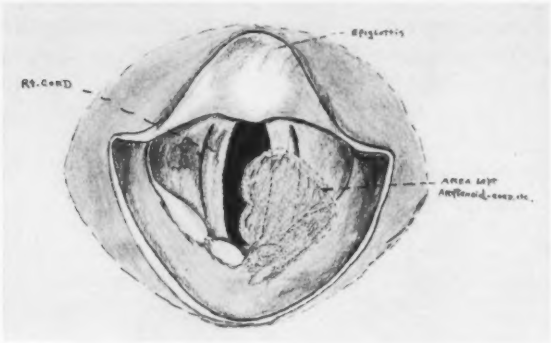

Fig. 2.

Looking at the throat you see a large, spotted, irregular, nonpulsating, reddish purple mass, slightly elevated at certain points, extending over the posterior two-thirds of the palate and involving the entire uvula. The discoloration of certain sections varies from dark red to dark purple. The mass extends down the anterior and posterior faucial pillars. On both sides of the posterior pharyngeal wall, extending downward into the pharynx and upward behind the palate, may be seen several large areas with similar discoloration resembling dilated vessels. The area on the palate is slightly sensitive to touch and can be compressed but shows no pulsation (see Fig. 2).

Indirect examination of the larynx shows extension of the growth to base of tongue, epiglottis, right cord and right arytenoid. On the left side, a mass, reddish purple in color, covers the left arytenoid and cord from the posterior commissure to within a few millimeters of the anterior commissure and extends beyond the midline. This mass, on admission to the hospital, was not as large as it is at the present time. The

increase in size is attributed to reaction from the radium (see Fig. 4).

This is a stereoscopic X-ray of the larynx made by the Roentgenologist, Dr. F. M. Law, showing the epiglottis and the thyroid gland quite distinctly. Here is the mass of tissue in the larynx just presented. This is a curious fact. The esophageal wall is ballooned out markedly below the laryngeal mass, and Dr. Law thought that there might possibly be some

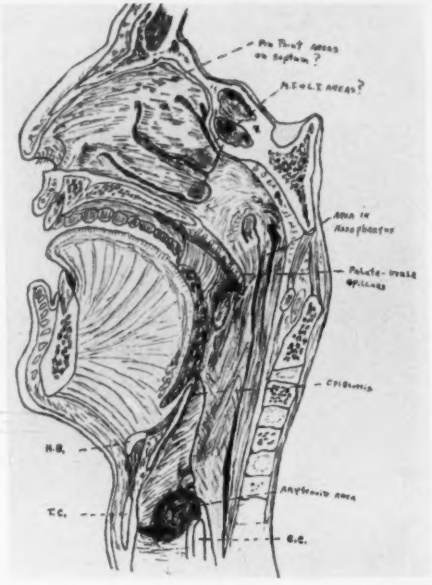

Fig. 3.

extension of the growth into the esophagus. This could explain the patient's dysphagia at intervals since he was 18 years of age. The involvement of the esophagus has been confirmed by esophagoscopic examination (see Fig. 5).

This is a microscopic section made by the pathologist, Dr. A. A. Eggston, of a biopsy taken from the uvula. This biopsy was removed by means of diathermy to prevent bleeding. Pathological report: Tissue shows thickened hyperkeratosis, squamous mucosa. The submucosa consists of trabeculae of

dense connective tissue surrounding dilated and cavernous blood spaces. There is some mononuclear inflammatory reaction, particularly of the mucous glands. An occasional lymph space may be seen. Diagnosis: Cavernous hemangioma.

After admission on Jan. 14, the patient received general care of the nose and throat; hot saline throat irrigations; ephedrin sulphate and carbolized oil in the nose. It was not necessary to pack the nose to check the bleeding.

The laboratory reports showed: Hemoglobin, 80 per cent; R.B.C., 4,350,000; W.B.C., 7,900. Differential: Polys., 37 per cent; S.L., 18 per cent; L.L., 8 per cent; eos., 1 per cent;

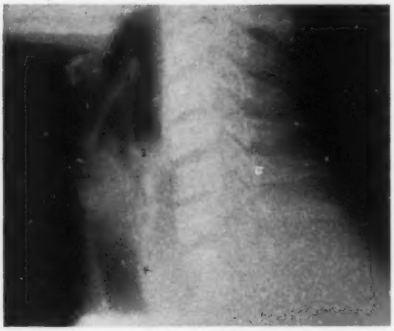

Fig. 4. 1. Epiglottis. 2. Thyroid gland. 3. Mass in larynx. 4. Ballooning out of esophageal wall.

platelets, 420,000; Wassermann: Negative; Kahn: Negative. Urinalysis: Negative for albumin and sugar. An occasional pus cell seen on microscopic examination. No casts.

X-ray report of the sinuses: There is a chronic pansinusitis with marked involvement of the frontals and antra. The ethmoidal labyrinth shows slightly less degree of involvement with edema and condensing osteitis. The sphenoidal cells show much less pathology. There is marked thickening of the mucous membranes of all the sinuses, but no polypoid degeneration. Stereoscopic films were made in an attempt to determine whether or not there was any extension of the growth into the sinuses. No evidences of this could be found.

X-ray report of the chest: Increase in vascular markings in region level with right middle lobe, indicating congestion, otherwise negative. A bronchoscopic examination was done by the bronchoscopist, Dr. D. H. Jones, on Jan. 18, which revealed nothing further than previously reported.

Patient was discharged to the clinic but was readmitted Jan. 28 with another hemorrhage from the throat. This hemorrhage ceased soon after the patient was admitted.

Dr. G. A. Robinson applied radium packs to either side of the neck in the region of the larynx as follows: Jan. 30, 1935:

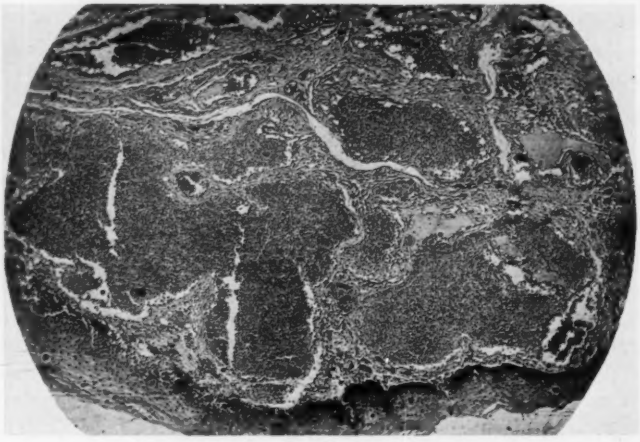

Fig. 5.

200 mgs. at 4 cm. distance 41 hours, 8200 mghrs; Feb. 8: 300 mgs. at 4 cm. distance 18 hours, 5400 mghrs; Feb. 14: 250 mgs. at 4 cm. distance 12 hours, 3000 mghrs.

The patient has had a total of 16,000 mghrs. He was discharged to the clinic on Feb. 16. His hoarseness has cleared up to a certain extent. After his discharge he had some slight bleeding from his throat on rising each morning, but this has ceased and he has had no further hemorrhages. There is no pain. There is a distinct radium reaction on either side of the neck, which you can see. He claims that he doesn't have to shave any more. On examining the lesions today, we find the

mass in the larynx about half its former size and the condition of the palate is clearing up remarkably well.

CONCLUSION.

1. Cavernous hemangiomas are congenital in origin although the growths may not manifest themselves for years. The age of cases reported ranges from a few months to 60 years.

2. These tumors of the nasopharynx and larynx are important clinically because: *a.* They are very prone to hemorrhage. *b.* In the nasopharynx they are liable to invade the sinuses, and are especially dangerous in the region of the ethmoidal labyrinth. *c.* Their invasion is by contiguity rather than by continuity.

3. They are not considered malignant and do not metastasize, although there have been a few malignant cases reported; however, they may be associated with sarcomas.

4. They may involve any part of the body with the exception, according to Kronmann, of the cornea and cartilages.

5. Highly vascular tumors, fibromas, bleeding polyps, etc., should never be classified as true hemangiomas.

6. The method of treatment in the majority of cases is through the use of radium packs, although in certain circumscribed cases, surgical removal is advocated; however, there is the liability to severe hemorrhage and the possibility of recurrence after surgical removal. They should be considered carefully as to location, extension, character of growth, clinical symptoms, etc., before treatment.

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THE TRIGEMINOFACIAL REFLEXES.*

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The otolaryngologist and ophthalmologist are often confronted with diseases in which the trigeminal and facial nerves are involved. Therefore, an understanding of the reflex mechanisms of these two nerves and the relationship of one to the other will be of aid in differentiating neurological from local involvements of a non-neurologic nature in disease of the head. Conditions such as sinus diseases, eyelid infections, sinus thrombosis, arthritis, injury, syphilis, influenza, diabetes and arteriosclerosis may affect the peripheral portions of one or both of these nerves and call for a thorough differential diagnosis between trigeminal neuralgia and any one of the above. Intracranial diseases and involvements of the upper portions of the spinal cord may, furthermore, be referred to as responsible for the disturbance in facial structure functioning.

In a previous paper[1], the writer dealt with the facial reflexes, considering mainly the reactions of the facial musculature to tapping various parts of the face in diseases affecting the peripheral portions of the facial nerve such as met with in Bell's palsy, or trauma and infection of the same in the course of otitis media and mastoid operations as well as in involvements of nuclear and supranuclear structures of the VIIth nerve. In an article dealing with the opticofacial reflex[2], it was furthermore shown that the afferent paths of the facial consisted not only of the trigeminal but also of the optic and acoustic nerves, these forming separate reflex arcs.

The case to be presented here will prove that by the term facial reflexes as commonly used in clinical examination we really mean trigeminofacial reflexes, since stimulation of the facial structures by tapping involves stimulation of the Vth nerve. It will be evident that not even in the Chvostek sign do we produce a pure motor stimulation of the facial musculature without involving the trigeminal component also.

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*From the Neurological Service of the City Hospital.

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That the sensory portion of the trigeminal is extensive, so that it comes in contact with the substantia gelatinosa of Rolando in the upper two cervical metameres, is well known. In his work on the craniocervical reflexes[3] the writer has emphasized upon the role that this nerve plays in not only cranial but also craniocervical reflexes. The reader is referred to that article for the neuroanatomy and neurophysiology of the afferent part of this reflex[4].

TECHNIQUE.

The technique of the elicitation of these reflexes is simple. Tapping in the zygomatic region as shown in Fig. 1 results in a drawing upward and outward of the upper lip of the same side. This is due to a contraction of the zygomaticus major and minor muscles. In Fig. 2 may be noted the method of eliciting the reaction of the levator labii superioris propruis and the alaequae nasae, as well as the reactions of some of the deeper facial muscles. For the reactions of the remaining muscles the reader is referred to the article on facial reflexes[5].

In the following case we find definite proof that the facial reflex reactions are due largely to the stimulation of the reflex arc described above.

E. B., an adult white male, age 49 years, was examined in the neurological wards of the City Hospital, where he was found complaining of loss of use of his right arm and wasting of the muscles in both of his hands. He related that his trouble began 15 years ago with pin and needle sensations in the right side of his head and face. This continued for about a year and was followed by his other complaints.

The past personal and family histories did not disclose anything that might be etiologically linked with his present condition.

Neurological examination showed no co-ordinative or marked motor disturbances. The reflex changes of his body and extremities were not remarkable. There was marked atrophy of the thenar and hypothenar eminences and of the interossei of both hands. The right forearm, arm and shoulder musculature also showed evidences of atrophy. There was definite pain and temperature dissociation on the right side. The

right side of the head, face, and tongue presented an anesthesia to all forms of sensation.

Tapping of the facial regions on the right side gave no response while when the left side was tapped even gently most of the muscles contracted, giving lively responses. Even the winking reaction was absent on the right side. When the stimulation of the right side of the face by tapping was intensified and limited to the muscle proper, *i.e*, the areas of origin and insertion being omitted, there was a very faint partial questionable reaction which was probably myotatic in nature. The diagnosis of this case was syringomyelia.

Fig. 1.

Fig. 2.

Fig. 1. Method of elicitation of zygomaticus major and minor reflexes.

Fig. 2. Method of elicitation of the levator labii superioris proprius and alaequae nasae reflexes.

Many cases of hemiplegia due to hemorrhage or encephalitis were studied and the trigeminofacial reflexes compared. These reactions were of particular value in differentiating many cases suffering from the former disease from those afflicted with the latter. In hemorrhage, as well as in other localized lesions of the brain, the facial reaction was usually unilateral, while in encephalitis and in diffuse lesions, the reaction to tapping one side of the face was a bilateral one. In diseases of the middle and posterior fossa, where the cranial nerves were involved, it was particularly noted that the differential diagnosis between brain abscess and a purulent basilar meningitis, for instance, could be more easily made with the help of the above reflex reactions.

CONCLUSIONS.

1. A case was presented in which an involvement of the Vth cranial nerve was accompanied by an absence of reaction of the facial muscles when the same side of the face was tapped.

2. The term facial reflex should therefore be changed to trigeminofacial reflex.

3. The trigeminofacial reflex reactions are of particular use in differentiating between localized and diffuse lesions of the brain.

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AUDITORY FUNCTION STUDIES IN AN UNSELECTED GROUP OF PUPILS AT THE CLARKE SCHOOL FOR THE DEAF:

I. GENERAL SURVEY OF HEARING ACUITY.*†

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Detailed studies of hearing acuity in pupils in schools for the deaf, based on audiometric tests carried out under controlled conditions and over long periods of time, should add to the knowledge of deafness in childhood and of auditory function in general; they should be of value in a more scientific adaptation of the educational program to the needs of the auditorially handicapped; and, coupled with audiometric studies of other members of the family, they should form the basis of programs for the prevention of future deafness. With these three aspects of the subject in mind, the study of auditory function has occupied an important place in the study of the deaf child at the Clarke School, in the hope that further knowledge may be acquired and eventually applied in these several fields.

Many of the studies which have been made in schools for the deaf in the past have failed to give a complete picture of the hearing acuity of such groups: (1) because of the impossibility of obtaining an accurate picture of the auditory function of a deaf child at a single test, if his sense of hearing has not been previously trained; (2) because of the inadequacy of the audiometers in use in most schools for the deaf to explore the entire auditory field; and (3) because many of the audiometric studies have been based on air conduction alone rather than on the results obtained by both air and bone conduction. It is hoped that some of these difficulties may be obviated in the audiometric studies which are now being made in various centers in this country and abroad. Among such investigations

*From the Research Department at the Clarke School for the Deaf.

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being carried on at the present time, mention should be made of those at Central Institute under Dr. Max A. Goldstein,[1] of those in the London County Schools for the Deaf under Dr. Phyllis M. T. Kerridge[2] of the University of London, of those in Manchester, under Dr. and Mrs. A. W. G. Ewing[3] of the University of Manchester. The extensive survey of pupils in public schools for the deaf in this country, carried out under Dr. George E. Shambaugh[4] and others for the National Research Council, assembled a large amount of valuable data on the subject which should serve as a foundation for future detailed studies of smaller groups.

At the Clarke School, the average pupil may be studied over a period of 10 years. A program[5] has been devised for the training and testing of hearing in the preschool or entering child, and emphasis is placed on the use of the child's residual hearing throughout his school years. Frequent audiometric tests by air and bone conduction are carried out at the time of the entrance of each pupil and regularly at six-month intervals or oftener thereafter; and an amplified audiometer is now available which extends the intensity range nearly to the "threshold of feeling" by air conduction.

In this general survey, we have attempted to establish the general trend of hearing acuity by air and bone conduction: (1) for an unselected group of pupils in a school for the deaf; and (2) for the principal etiological groups of which such a group of pupils is composed.

DATA AND TECHNIQUE.

The data analyzed consist of audiometric readings by air and bone conduction on 100 members of the upper and middle schools in the Fall of 1934. The pupils were unselected as to cause of deafness, character or degree of hearing loss, and may, therefore, be considered a fair sample of Clarke School's older pupils, ranging in age from 9 to 20 years. Boys constitute 46 per cent of the group, and girls, 54 per cent.

Tests were carried out in a sound-proof laboratory under controlled conditions. A morning period was selected, in order to eliminate the effects of general fatigue and of auditory stimulation to the greatest possible extent. The amplified 2-A audiometer* was used throughout this study; the advantages

*We are indebted to Dr. Harvey Fletcher, of the Bell Telephone Laboratories, for the modifications in the 2-A Western Electric audiometer, which extend the intensity range 25 db. at each octave, and which render possible readings at quarter-octave intervals.

of its increased intensity range may be seen in Charts 1 and 2. A hearing curve has been obtained from even the most profoundly deaf pupils, some of whom gave no response whatsoever on the unamplified 2-A audiometer. Readings were made by both air and bone conduction at octave intervals from 64 to 8192 d.v., inclusive, and the same threshold was obtained on at least three successive readings before the result was recorded. Hearing by bone conduction has been tested on the amplified 2-A audiometer by means of the Western Electric bone conduction receiver placed over the mastoid region. The limit of intensity of the amplified 2-A audiometer is so near the line of "total loss of serviceable hearing" or "threshold of

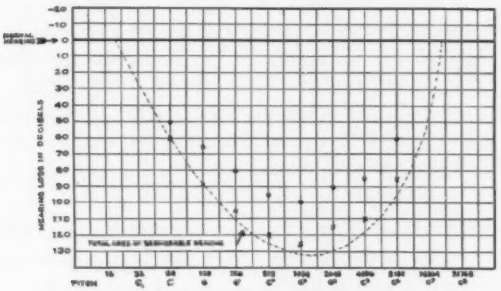

Chart 1. Audiogram showing intensity limits of the original 2-A audiometer by dots, of the amplified 2-A audiometer by crosses.

feeling" by air conduction that an arbitrary score* of total loss has been given to *no response* at maximum intensity in all computations in this study. Since our analysis showed that means for the right, left, better and poorer ears were similar, with the exception of the ear, nose and throat group, we have presented an analysis of the threshold readings for the *right ear only* in this general survey of hearing acuity.

GENERAL TREND OF HEARING ACUITY BY AIR AND BONE CONDUCTION FOR THE ENTIRE DEAF GROUP.

In order to determine the general trend of hearing acuity for the entire group, mean hearing curves by air and bone

*The arbitrary scores given to *no response* at maximum intensity on the amplified 2-A audiometer are: 60 at 64 d.v.; 90 at 128 d.v.; 110 at 256 d.v.; 125 at 512 d.v.; 130 at 1024 d.v.; 130 at 2048 d.v.; 120 at 4096 d.v.; and 95 at 8192 d.v.

conduction have been established, with the frequency distribution at each pitch.*

The mean hearing curve by air conduction, as shown in Chart 3, descends gently from 64 d.v. to 4096 d.v.; at 8192 d.v. the mean is outside the limit of intensity of the amplified 2-A audiometer. Hence, the mean curve drops off at 4096 d.v., the remainder of the curve being indicated by a dotted line. From the direction of the curve, it is evident that the hearing loss tends to increase from the low to the high tones. In computing

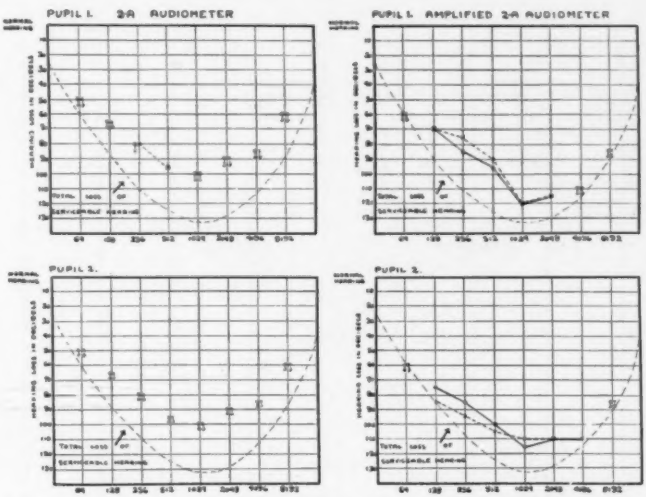

Chart 2. Audiograms showing advantages of the amplified 2-A audiometer.

the means and standard deviations, arbitrary scores† for total loss were given to *no response* at the maximum intensity on the amplified 2-A audiometer. The figure at the lower end of the frequency distribution at each of the eight pitches in Chart 3 and subsequent charts indicates the number of pupils giving a response at each pitch, the difference between this figure and 100 being the number arbitrarily given a score for total loss at each particular pitch.

*We wish to acknowledge the advice and assistance of Dr. E. M. Jellinek, Director of the Biometric Department at the Worcester State Hospital, on the statistical methods used throughout this study.

†See footnote on page 48.

From the frequency distribution at each pitch in Chart 3, it is evident that an average group of unselected pupils in a school for the deaf is far from homogeneous from the standpoint of degree and character of hearing loss. This same fact is evidenced by the constants of frequency distribution by air conduction given in Table I, where one may see at a glance the wide range between the minimum and maximum readings at each pitch. Therefore, the shape of the mean curve itself is representative, but the scatter around the means is so great that the precise level of the curve is not absolutely established.

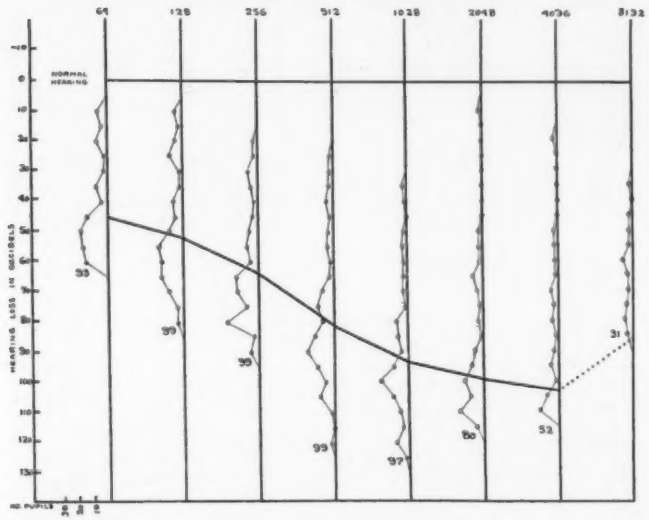

Chart 3. Mean hearing curve by air conduction for 100 unselected pupils, with frequency distribution at each pitch.

This lack of homogeneity in an average group of pupils in a school for the deaf emphasizes the need of an educational classification on the basis of amount of residual hearing.[6] This point will be considered in greater detail in a subsequent paper in this series dealing with the classification of audiograms.

Again referring to Chart 3, it is evident that there is the widest scatter, as one would expect, for the lower pitches, the amount of variability tending, in general, to decrease, while the amount of hearing loss tends to increase, from the low to

the high tones. In other words, the group, as a whole, have more residual hearing for the lower tones than for the higher, but there is much greater variation between individual readings for the lower tones than for the higher.

The mean hearing curve by bone conduction (see Chart 4) descends gently to 256 d.v., then more steeply to 1024 d.v., and at the three higher pitches the means are outside the limit of intensity of the amplified 2-A audiometer. The bone conduction mean curve, therefore, descends from left to right and drops off at 1024 d.v., the remainder of the curve being indicated by a dotted line. The dotted line represents the highest possible arbitrary level. If greater amplification by bone conduction were possible, a larger number of responses

	64 d.v.				128 d.v.				256 d.v.			
Entire Group	Mean	S. D.	Min.	Max.	Mean	S. D.	Min.	Max.	Mean	S. D.	Min.	Max.
Air conduction	43.9	14.7	10	60*	51.5	18.2	10	90*	63.7	18.1	20	110*
Bone conduction	8.9	5.7	-15	25	13.9	7.9	-10	35	26.2	9.4	-10	45

	512 d.v.				1024 d.v.				2048 d.v.			
Entire Group	Mean	S. D.	Min.	Max.	Mean	S. D.	Min.	Max.	Mean	S. D.	Min.	Max.
Air conduction	80.8	20.4	25	125*	93.9	18.1	35	130*	99.4	23.8	10	130*
Bone conduction	48.5	12.5	0	70*	73.8	12.9	15	85*	77.5*	13.2	5	85*

	4096 d.v.				8192 d.v.			
Entire Group	Mean	S. D.	Min.	Max.	Mean	S. D.	Min.	Max.
Air conduction	103.1	21.2	20	120*	86.6*	14.2	35	95*
Bone conduction	80.9*	7.9	20	85*	68.8*	7.6	15	70*

Table I. Standard deviation and constants of frequency distribution, including mean, minimum and maximum readings, at each pitch, by air and bone conduction, for an unselected group of 100 pupils.

*Outside limit of intensity of amplified 2-A audiometer.

might be obtained at the higher frequencies, thus lowering the level of the curve represented by the dotted line. The means, as obtained on the amplified 2-A audiometer, have been plotted with reference to the line of normal hearing, as recommended by Dr. Fowler, Dr. Fletcher, and others. In computing the

means, the same arbitrary values* were given to no response at maximum intensity as were given to *no response* by air conduction; this is a purely arbitrary procedure for the bone conduction data at the present time, as the "threshold of feeling" or "total loss of serviceable hearing" has not yet been established for bone conducted sounds. The trend of the mean curve is valid, being based on readings on 100 representative individuals. The level of the individual points is also reliable, as the scatter around the means is not great (see Chart 4 and Table I).

From the frequency distributions in Charts 3 and 4, it would appear that the entire group is much more homogeneous from the standpoint of bone conduction than from that of air conduction.

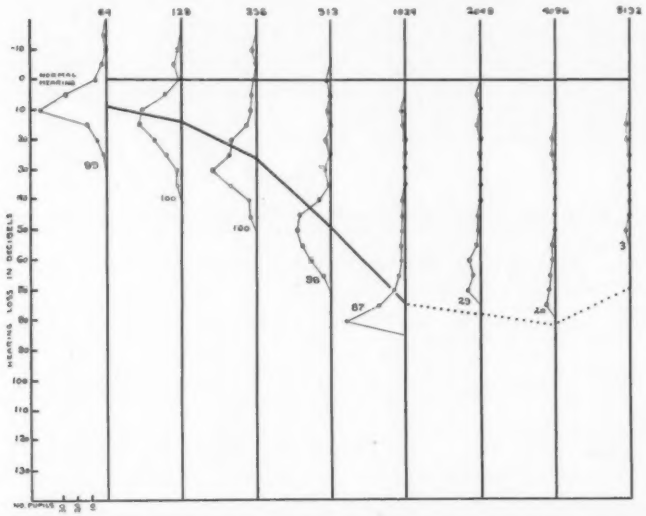

Chart 4. Mean hearing curve by bone conduction for 100 unselected pupils, with frequency distribution at each pitch.

In Chart 5, it is at once apparent that the mean bone conduction threshold is nearer normal at the lower and middle frequencies than the mean air conduction threshold. At the three higher frequencies, the bone conduction means are

*See footnote on page 48.

outside the intensity range of the amplified 2-A audiometer, while the air conduction mean curve does not drop off until 4096 d.v. A larger number give *no response* at maximum intensity by bone conduction than by air conduction at the higher frequencies, as shown in Chart 6. In other words, the mean hearing loss tends to increase more rapidly from the low to the high tones, with a larger proportion of *no responses* for the three highest tones, by bone conduction than by air conduction.

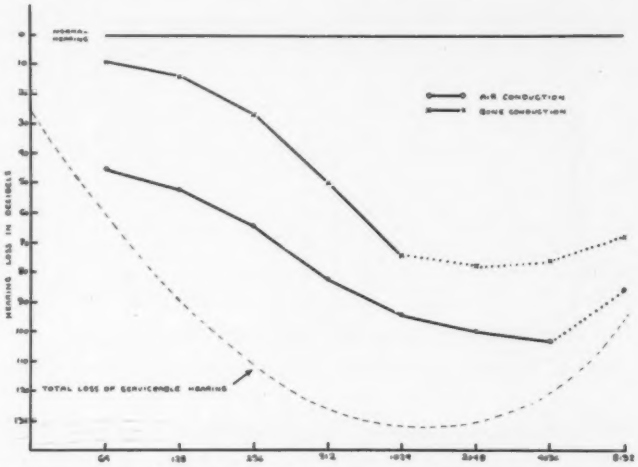

Chart 5. Mean hearing curves by air and bone conduction for 100 unselected pupils.

The practical significance of this difference in the intensity and the frequency range by air and bone conduction we cannot evaluate absolutely until the "threshold of feeling" for bone conducted sounds is established, and until we have correlated the above findings with the results of speech-sound tests in pupils who have had ample opportunities to become familiar with both types of amplification. It would seem probable that individuals giving *no response* by bone conduction to these higher tones at maximum intensity on the amplified 2-A audiometer have so little residual hearing at these frequencies that it would be of no practical value for the interpretation of speech sounds by bone conduction. It would, therefore, seem probable that the pupil whose air conduction curve goes

through the higher frequencies, but whose bone conduction curve drops off at 512 or 1024 d.v., should receive more help by air conduction amplification, because of the wider range over the speech-sound frequencies (400-8000 d.v.). These pupils should receive help in the actual discrimination of speech sounds and in the interpretation of speech by air conduction amplification. Again, it would seem probable that the profoundly deaf child, whose audiogram drops off at 512 or 1024 d.v. by both air and bone conduction, should receive

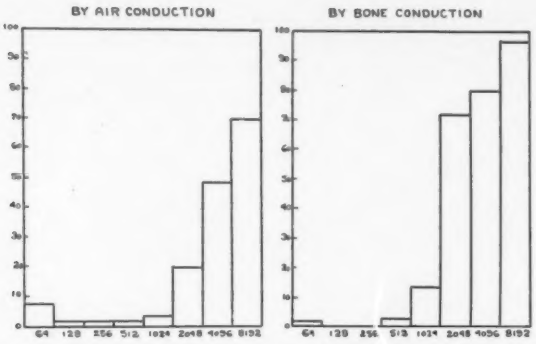

Chart 6. Number of *no responses* at maximum intensity at each pitch.

greater help by bone conduction amplification because the bone conduction threshold is much nearer normal at these lower frequencies. In the latter case, the aid will assist the pupil in accent, inflection and phrasing, but not in discrimination of speech sounds.

GENERAL TREND OF HEARING ACUITY BY AIR AND BONE CONDUCTION FOR THE PRINCIPAL ETIOLOGICAL GROUPS.

The etiological distribution of the pupils included in this study is as follows: deafness of congenital origin, 50 per cent; deafness of probably congenital origin, 16 per cent; deafness due to cerebrospinal meningitis, 14 per cent; deafness due to primary disease of the ear, nose and throat, 12 per cent; and deafness due to a toxic neuritis of the auditory nerve following an acute infectious disease, 8 per cent (see Table II). This distribution is typical of Clarke School's student population during the past five years. We are accustomed to classify as

"probably congenital" deafness recognized prior to the acquisition of speech when both parents and physicians are in doubt as to whether it has been present since birth or has resulted from some slight illness during infancy.

Cause of Deafness	Number of Pupils	Percentage
Congenital	50	50
Probably congenital	16	16
Cerebrospinal meningitis	14	14
Disease of ear, nose and throat	12	12
Toxic neuritis	8	8
Total	100	100

Table II. Etiological classification of 100 unselected pupils included in this study.

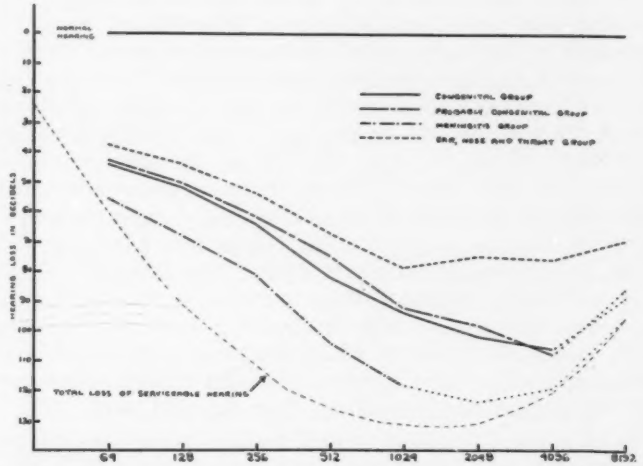

Chart 7. Mean hearing curves by air conduction for principal etiological groups.

In Chart 7, mean hearing curves by air conduction for the four largest etiological groups have been plotted. It will be noted that the mean curve by air conduction for the ear, nose and throat group is considerably higher than those for the other etiological groups; it descends gradually to 1024 d.v., and then tends to remain horizontal, except for a slight rise from 1024 to 2048 d.v. and from 4096 to 8192 d.v. The mean curves for the congenital and probably congenital groups descend at practically the same level to 4096 d.v.; at 8192 d.v.

the means for both groups are outside the intensity range of the amplified 2-A audiometer, the remainder of the curves being indicated by dotted lines. Therefore, the mean curves for both groups descend from left to right and drop off at 4096 d.v. At all pitches their level is considerably above that for the meningitis group. The mean curve for the meningitis group is by far the lowest of the four; it descends to 1024 d.v., and at the three higher pitches the means are outside the limit of intensity of the amplified 2-A. Therefore, the mean curve for the meningitis group descends from left to right and drops off at 1024 d.v. Mean hearing curves have not been plotted for the toxic group because of the small number of individuals included in the group.

The mean hearing loss, the range, and the standard deviation for each group may be seen in Table III, in which the constants of frequency distribution are given in the form of the mean, minimum and maximum readings. It is apparent that we have the widest scatter in the ear, nose and throat group. This is as one would expect on the basis of the knowledge which we have concerning the after-effects of disease of the ear, nose and throat. In the group are individuals with chronic suppurative otitis media of long duration; in some the damage to the auditory apparatus has been very great, in others, comparatively slight. The same is true for those who have had mastoiditis; some belong in a profoundly deaf class and some would be classified as hard-of-hearing only. Hence, the group in which deafness is due to primary disease of the ear, nose and throat is a far from homogeneous one, the graphs varying in both character and level.

There is also a wide variation in the congenital and probably congenital groups, though somewhat less than in the ear, nose and throat group. This, again, is as one would expect. The fact that a child is born with defective auditory function indicates nothing as to the particular part of the auditory apparatus which may be lacking, defective or damaged by disease, or the degree of the damage or defective development. It should also be borne in mind that secondary disease may be superimposed upon the primary congenital defect. Hence, in the congenital and probably congenital groups, one may expect any type of graph at any level. From our functional studies up to date, it seems evident that the congenital and probably congenital groups are in no sense of the word homogeneous

from the standpoint of underlying pathology and resulting functional impairment. In order eventually to resolve these groups into their homogeneous parts, detailed functional

	64 d.v.				128 d.v.				256 d.v.			
Cause of Deafness	Mean	S. D.	Min.	Max.	Mean	S. D.	Min.	Max.	Mean	S. D.	Min.	Max.
Congenital	43.4	14.0	10	60	51.3	16.1	15	80	63.3	15.9	30	90
Probably congenital	42.8	15.7	10	60	50.0	19.3	10	75	61.2	20.2	20	85
Cerebrospinal meningitis	55.4	6.1	45	60	66.1	9.1	55	90*	80.4	1.3	65	110*
Disease of ear, nose and throat	37.1	21.8	10	60	42.1	24.9	10	80	52.9	24.9	20	90
Toxic neuritis	38.7		20	55	43.7		25	65	58.1		45	70

	512 d.v.				1024 d.v.				2048 d.v.			
Cause of Deafness	Mean	S. D.	Min.	Max.	Mean	S. D.	Min.	Max.	Mean	S. D.	Min.	Max.
Congenital	80.9	16.8	35	105	93.1	7.9	35	120	100.7	19.3	50	130*
Probably congenital	74.7	19.2	30	95	91.6	13.9	55	120	96.9	18.1	65	130*
Cerebrospinal meningitis	103.2	9.4	90	125*	117.9	10.1	95	130*	122.1*	13.1	95	130*
Disease of ear, nose and throat	65.4	25.5	25	100	76.7	28.8	35	115	74.2	28.5	10	130*
Toxic neuritis	76.2		55	105	88.1		60	120	94.3		60	130*

	4096 d.v.				8192 d.v.			
Cause of Deafness	Mean	S. D.	Min.	Max.	Mean	S. D.	Min.	Max.
Congenital	105.0	17.9	55	120*	88.8*	11.0	50	95*
Probably congenital	106.9	11.4	70	120*	86.6*	12.5	60	95*
Cerebrospinal meningitis	117.1*	7.2	110	120*	95.0*		95*	95*
Disease of ear, nose and throat	75.4	31.1	20	120*	68.7	20.4	35	95*
Toxic neuritis	100.6		70	120*	85.0*		60	95*

Table III. Standard deviation and constants of frequency distribution, including mean, minimum and maximum readings at each pitch, by air conduction, for the principal etiological groups.

*Outside limit of intensity of amplified 2-A audiometer.

studies over a period of years on a large number of congenitally deaf individuals must be linked up to detailed postmortem studies. It is hoped that this will be one of the results of the research which is already under way in the various centers.

Finally, we come to the group in which deafness is due to cerebrospinal meningitis, which is the most homogeneous group in a school for the deaf. As is readily seen in Table III, the variation is strikingly less than in any other group. The child is always profoundly deaf, as is evident from the mean curve.

As previously stated, this survey is based on statistical analyses of threshold readings for the right ear only. In Chart 8, however, mean curves have been plotted for the right,

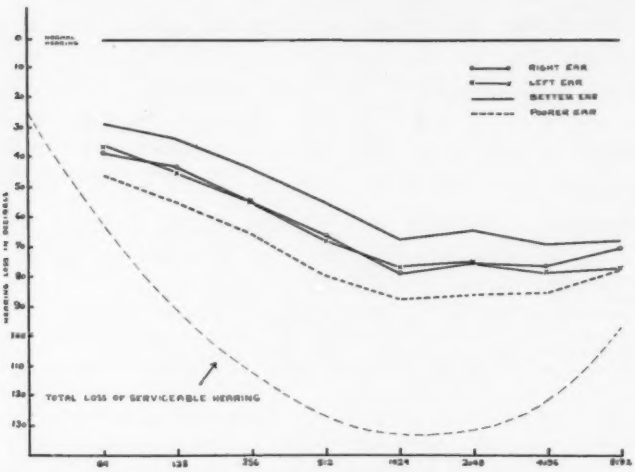

Chart 8. Mean hearing curves by air conduction for right, left, better and poorer ears of the ear, nose and throat group.

left, better and poorer ears for the ear, nose and throat group, in which the two ears are sometimes quite unequally affected. It will be seen that the mean curves for the right and the left ears are very similar, while the mean curve for the better ear is considerably higher than that for the poorer ear at all pitches.

In Chart 9, mean hearing curves by bone conduction for the same etiological groups have been plotted with reference to the line of normal hearing. It is at once apparent that the order of the level of the mean curves is similar to that of the mean air conduction curves, but that the mean bone conduc-

tion curves are not nearly so widely separated, the means for the congenital and probably congenital groups being at only a slightly higher level than the means for the meningitis group. The mean curve by bone conduction for the ear, nose and throat group descends gently to 256 d.v., abruptly to 1024 d.v., remains horizontal to 2048 d.v., and then again descends slightly to 4096 d.v.; at 8192 d.v., the mean is outside the limit of intensity of the amplified 2-A audiometer. The mean curves by bone conduction for the congenital and probably congenital groups descend more abruptly to 1024 d.v., where they drop

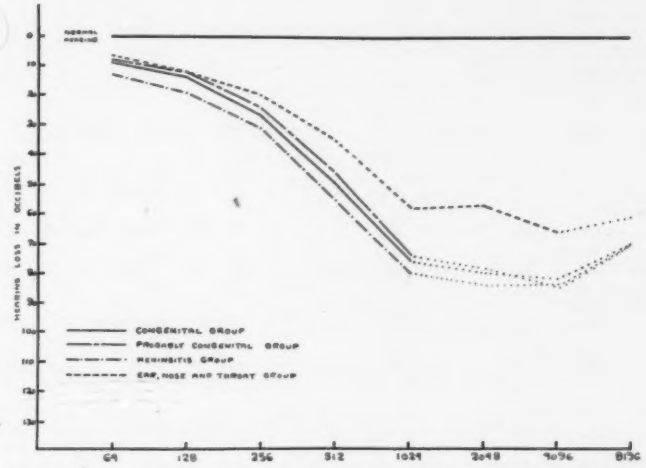

Chart 9. Mean hearing curves by bone conduction for principal etiological groups.

off, the means for the three highest tones being outside the limit of intensity of the amplified 2-A audiometer. The mean curve by bone conduction for the meningitis group also descends abruptly, and drops off at the same octave, namely at 1024 d.v.

The mean hearing loss, the range and standard deviation for each of the etiological groups, by bone conduction, may be seen in Table IV. The etiological groups, like the entire group, are more homogeneous from the standpoint of bone conduction than from that of air conduction, the difference being least marked in the meningitis group, because in that group the amount of variability is small by both air and bone conduction.

In Chart 10, the mean curves by air and bone conduction for each of the principal etiological groups may be compared with the line of normal hearing, bearing in mind constantly the

	64 d.v.				128 d.v.				256 d.v.			
Cause of Deafness	Mean	S. D.	Min.	Max.	Mean	S. D.	Min.	Max.	Mean	S. D.	Min.	Max.
Congenital	8.6	4.9	-15	20	13.7	7.6	-10	35	26.6	7.9	5	45
Probably congenital	7.8	7.1	0	25	11.9	8.5	- 5	35	24.4	8.6	5	40
Cerebrospinal meningitis	12.8	5.2	5	25	19.2	4.9	10	25	31.1	7.1	20	45
Disease of ear, nose and throat	6.7	6.2	- 5	20	11.7	9.2	- 5	25	20.0	14.3	-10	40
Toxic neuritis	9.4		5	15	13.1		0	25	28.8		10	35

	512 d.v.				1024 d.v.				2048 d.v.			
Cause of Deafness	Mean	S. D.	Min.	Max.	Mean	S. D.	Min.	Max.	Mean	S. D.	Min.	Max.
Congenital	50.1	8.2	30	70*	75.6	8.8	45	85*	80.0*	9.7	45	85*
Probably congenital	46.3	10.8	20	65	74.1	6.7	65	80	79.4*	8.6	60	85*
Cerebrospinal meningitis	55.4	6.4	45	65	80.0	5.7	70	85*	83.9*	3.9	70	85*
Disease of ear, nose and throat	35.0	21.0	0	65	58.3	22.4	15	80	56.7	24.0	5	85*
Toxic neuritis	51.3		20	65	75.0		40	85*	79.3*		55	85*

	4096 d.v.				8192 d.v.			
Cause of Deafness	Mean	S. D.	Min.	Max.	Mean	S. D.	Min.	Max.
Congenital	82.1*	6.8	65	85*	69.6*	2.8	50	70*
Probably congenital	83.8*	3.3	75	85*	70.0*		70*	70*
Cerebrospinal meningitis	85.0*		85*	85*	70.0*		70*	70*
Disease of ear, nose and throat	66.3	22.0	20	85*	61.3*	19.6	15	70*
Toxic neuritis	81.0*		70	85*	70.0*		70*	70*

Table IV. Standard deviation and constants of frequency distribution, including mean, minimum and maximum readings at each pitch, by bone conduction, for the principal etiological groups.

*Outside limit of intensity of amplified 2-A audiometer.

arbitrary values assigned to *no response*. For the separate etiological groups, as for the entire group (see Chart 5), the mean bone conduction threshold is nearer normal at the

lower and middle frequencies, than the mean air conduction threshold, the difference being least marked in the ear, nose and throat group, in which there is the widest frequency range by bone conduction (see Chart 10). There is a wider frequency range by air conduction for all groups with the exception of the meningitis group, in which the mean curves by both air and bone conduction drop off at 1024 d.v.

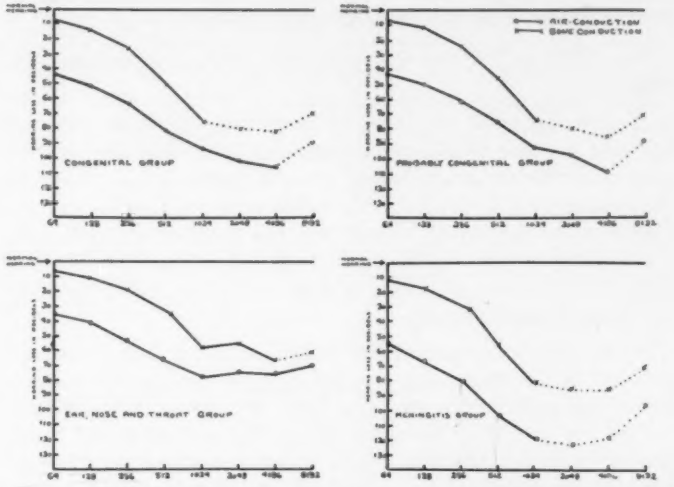

Chart 10. Mean hearing curves by air and bone conduction for principal etiological groups.

COMMENTS AND SUMMARY.

In the first of a series of auditory function studies, we have sought to establish the general trend of hearing acuity, by air and bone conduction, for an average group of 100 pupils in a school for the deaf, and for the principal etiological groups of which such an average group is composed.

The findings for the entire group indicate that:

1. By air conduction, the mean hearing loss increases, while the amount of variability between individual readings decreases, from the low to the high tones.

2. By air conduction, the group is far from homogeneous from the standpoint of type and degree of hearing loss.

3. The amount of variability between individual readings at all pitches is much greater by air conduction than by bone conduction.

4. By bone conduction, the average threshold is nearer normal at the lower and middle frequencies than the mean air conduction threshold; at the three higher frequencies, however, the bone conduction means are outside the intensity range of the amplified 2-A audiometer, while the mean air conduction curve does not drop off until 4096 d.v.

The findings for the four principal etiological groups indicate that:

1. The order of the level of the mean curves is the same by both air and bone conduction: the threshold for the ear, nose and throat group being nearest normal, and those for the congenital, probably congenital and meningitis groups following in the order named, but the mean bone conduction curves are not so widely separated, the means for the congenital groups being at only a slightly higher level than those for the meningitis group.

2. By air conduction, the mean hearing loss tends, in general, to increase and the amount of variability between individual readings to decrease from the low to the high tones for all groups, with the exception of the ear, nose and throat group, in which the loss tends to remain stationary beyond 1024 d.v. and the amount of variability to remain large.

3. By air conduction, the frequency range is greatest for the ear, nose and throat group, while the mean curves for the congenital and probably congenital groups drop off at 4096 d.v., and for the meningitis group at 1024 d.v.

4. By bone conduction, the average threshold is nearer normal at the lower and middle frequencies than the mean air conduction threshold for all groups; however, there is a wider frequency range by air conduction than by bone conduction for all groups with the exception of the meningitis group, in which the mean curves drop off at 1024 d.v. by both air and bone conduction.

5. There is the widest scatter, by air conduction, in the ear, nose and throat group, the next widest in the congenital and probably congenital groups, and the smallest in the meningitis group.

The educational significance of these findings is as follows:

1. The great lack of homogeneity from the standpoint of type and degree of hearing loss emphasizes the need of an educational classification in schools for the deaf on the basis of detailed auditory function studies, as well as according to mental achievement. The value of such studies, as a basis for classification, will be considered in the second paper in this series.

2. The differences in intensity and frequency range by air and bone conduction are of importance in the selection of type of amplification. The practical significance of these differences will be further studied in a later investigation in which audiometric findings by air and bone conduction will be correlated with speech sounds heard with the unaided ear and with both types of amplification.

3. The application of audiometric findings to the educational program for the deaf child calls for the co-operation of otologists, research workers, boards of education and educators.

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Clarke School for the Deaf.

VERTIGO.*

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Vertigo or dizziness is a complex problem. Oftentimes one is confronted with a difficult case and thereby placed in a state of mental confusion. Unless there is some co-ordinated and chronologic procedure of examination, an interesting problem may become delegated to the realms of mysticism.

To evaluate the full significance of vertigo one must understand its *modus operandi*, and this necessitates an anatomico-physiological explanation. Realizing the didactic boredom of lengthy anatomical dissertations, a very brief and practical elucidation would suffice.

Equilibrium is maintained by three factors: *1.* The labyrinth; *2.* the eye; *3.* the kinesthetic apparatus, *viz.*, muscles, tendons and arthroidal joints, etc. (Jones[1]).

The labyrinth is composed of three parts (see Diagram 1). The anterior part or the cochlear is the organ of hearing and does not concern us in this topic. The posterior part, or the three semicircular canals, has a kinetic function; that is, it analyzes the different motions of the body. The central part of the labyrinth contains two sacs: the utricle and saccule, and has a static function; namely, analysis of the different positions of our body in relation to space.

Histologically, the end organs of the labyrinth are the same. The organ of Corti for the cochlea, the maculae for the utricle and saccule, and the cristae acusticae of the three semicircular canals. These end organs are at right angles to each other, and have an antagonistic action so that the whole body is in perfect equilibrium.

The vestibular nerve emerges from the hair cells of the maculae of the utricle and saccule, and from the cristae acusticae of the semicircular canals, enters Scarpa's ganglion in the internal auditory meatus, where it unites with the

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cochlear nerve to form the auditory nerve, within one neurilemma. Then it passes toward the brain stem, where it divides again into its two constituent branches.

The vestibular nerve now enters the floor of the fourth ventricle. The majority of fibres enter Deiter's nucleus (see Diagrams 2 and 3), and from here to the posterior longitudinal bundle, which has connections with the oculomotor nuclei, producing the tract for nystagmus. This bundle, though highly important, does not concern us here. Some of the vestibular fibres enter Bechterew nucleus, and then pass through the in-

Fig. 1. Membranous labyrinth after Schaefer. (1) Anterior vertical semicircular canal. (2) Posterior vertical canal. (3) Horizontal semicircular canal. (4) Crus communis. (5) Crista acoustica of anterior vertical canal. (6) Crista acoustica of posterior vertical canal. (7) Crista acoustica of horizontal canal. (8) Utricle (macula). (9) Saccule (macula). (10) Utricle saccule canal. (11) Canalis reuniens. (12) Aqueductus vestibuli. (13) Saccus endolymphaticus blind pouch. (14) Cochlear canal.

ferior cerebellar peduncle to the vestibulocerebello nuclei; namely, emboliformis, globossus and fastigii. The fibres from the vertical canals shun the vestibulonuclei, go upwards, lateral to the midline to the level of middle of Pons, divide into two tracts, the nystagmic tract and the vertiginous tract. The latter passes through the middle cerebellar peduncle to the vestibulocerebellar nuclei. Here the vertiginous tracts unite as one and pass through the superior cerebellar peduncle, through the crus cerebrum to the opposite side of the brain. The exact location is not known. Mills claims it to be in the second temporal convolution, while others think it to be in the parietal lobe. However, this centre receives the terminal impulses of the vestibular nerve and by associatory fibres

relays them to the frontal lobe where the perception of motion is perceived and analyzed.

The remaining vestibular fibres enter the nucleus triangularis and then pass downward to the spinal cord, forming connections with the vagus nucleus, and then to the motor cells of the anterior horns of the spinal cord with the phrenic nerve. This explains the vegetative reactions; namely, nausea, vomiting, pallor, etc., associated with vertigo and nystagmus.

From this brief description it is observed that vertigo has a cerebral function. It may result from an irritation or disturbance of co-ordination, from any part of the vestibular ramification, starting from the end organ and terminating in its cortical centre. Philogenetically and ontogenetically, the auditory nerve is the last to develop of the cranial nerves. The cochlear portion is far the younger. This readily explains why this nerve is easily susceptible to toxins.

Patients devoid of or with destroyed labyrinth do not suffer vertigo. Typical example is the deaf mute. These patients maintain their equilibrium by the marked development of their ocular and kinesthetic senses. If these patients were to enter a dark room, or in any such circumstances where they cannot use their ocular apparatus, their equilibrium would be markedly disturbed and thereby develop inco-ordination (Dawson[2]).

The tabetic person depends upon his ocular and kinetic static senses. Loss of either one would result in inco-ordination.

The blind individual relies totally on his kinesthetic and kinetic static senses, and failure of either one eventuates in disequilibration.

Therefore, the maintenance of equilibrium depends upon at least two of the three senses; namely, kinetic static, ocular and kinesthetic. If any two are absent, inco-ordination is bound to ensue.

Now, it can be said that vertigo is a false impression of equilibrium or orientation of ourselves in relation to motion and space. According to Brunner there are two kinds of vertigo:

1. Systematized or true-turning dizziness.

2. Tactile or unsystematized dizziness: *a.* Errors in senation; *b.* lateropulsion of Babinski.

In true-turning dizziness, the objects turn in one direction, usually horizontal, either to the right or to the left, or the patient feels himself turning either to the right or to the left. Spontaneous true-turning dizziness is always associated with spontaneous nystagmus, and the direction of both is the same, and the patient always falls in the opposite direction. This is true of lesions in the end organ; namely, the labyrinth. However, we do know that multiple sclerosis can give this form of dizziness, and yet the labyrinth is perfectly intact. The central vestibular tract is invaded with formation of plaques (Grove[4]).

Tactile dizziness is due to disturbance in the muscle sense of the body. The labyrinth has a tonic influence on the skeletal musculature of the body. This type is subdivided into two forms:

a. Errors in sensation: Feels like he is drunk. In bed he feels as though he lies upside down, and, elevating his foot, thinks the level ground is like a hill. This is not accompanied by nystagmus, but sometimes found in labyrinth disease.

b. Lateropulsion of Babinski: Feels as if suddenly pushed or pulled to either right or left, but not back or forward. This comes in attack, and varies in degree. This occurs in nonsuppurative lesions of central vestibular system. *1.* Disseminated sclerosis. *2.* Encephalitis. *3.* Hydrocephalus. *4.* Tumors.

It is readily seen from the foregoing that this form of classification is not of any clinical value. We cannot definitely say that true-turning dizziness is indicative of a peripheral lesion, and not associated with a central lesion and *vice versa* for tactile dizziness.

If vertigo is analyzed with the clinical picture presented, and not *per se*, then this complex problem would automatically unravel itself. Clinically, vertigo is associated with four classes of lesions: *I.* Vertigo associated with aural lesions. *II.* Vertigo associated with C.N.S. lesions. *III.* Vertigo associated with ocular lesions. *IV.* Vertigo associated with systemic lesions.

I. VERTIGO ASSOCIATED WITH AURAL LESIONS:

It was Sir William Gowers who said that 90 per cent of cases of vertigo are due to aural lesions. This figure is not surprising since we know that vertigo must result from some disturbance connected with the ear. Vertigo is primarily an ear problem.

The pathological etiologic factors for vertigo within the ear and its surroundings are: *1.* Inflammation; *2.* hyperemia; *3.* ischaemia; *4.* pressure; *5.* faulty water metabolism; *6.* reflex phenomenon. These will be further dilated upon in conjunction with their lesions.

1. External Auditory Meatus: Any condition in canal that will irritate the drum will elicit a sympathetic reaction resulting in vertigo. Example: Foreign bodies, wax, external otitis, etc. The labyrinthine circulation is greatly influenced by the sympathetic system. Portmann,[3] of Bordeau, found on experimentation that paroxysmal vertigo can be produced either by

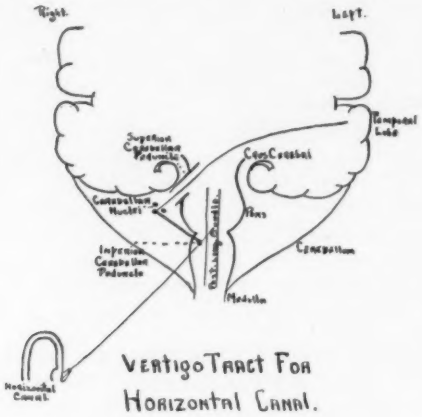

Fig. 2.

stasis and edema or by ischemia in the cochlea and vestibular branches of auditory artery by vasoconstrictors and vasodilators, and either condition produces vertigo.

2. Middle Ear: A. Suppurative Type: Neville[5] and Thomas.[6]

a. Acute suppurative condition without rupture may cause vertigo at times associated with nystagmus. This is elicited by pressure on the labyrinth, or through inflammatory process causing a perilabyrinthitis. Paracentesis is sufficient to relieve this condition.

b. Vertigo associated with a running ear is extremely dangerous to life. Danger of intracranial complication, especially associated with cholesteomata, is imminent.

c. Oftentimes in chronic cases with large perforations and in cases after radical mastoid, vertigo is not infrequently observed. This is induced in the same manner as insufflating the ear with cold or hot air.

d. Chronic running ear associated with intermittent attacks of vertigo not infrequently will reveal a fistula in the horizontal canal when examined.

e. Granulations or polypus with a running ear associated with vertigo call for complete and careful labyrinthine tests before same are removed.

B. Nonsuppurative Type: Aural vertigo associated with nonsuppurative type is not dangerous to life but may be very annoying.

a. Ankylosis of ossicles causes an increase in the intralabyrinth pressure, thereby disturbing the balance between the two labyrinths. Dundas Grant describes several cases where he removed the fixed ossicles and the vertigo disappeared.

b. Chronic catarrhal otitis is perhaps the greatest cause of vertigo. The recurrent attacks of vertigo are always associated with inefficiency of the Eustachian tube mechanism (Scott[7]). The closure of the tube causes an increased intratympanic pressure, which drives the stapes into the vestibule, causing an increase in intralabyrinthine pressure, thereby producing vertigo. He further observed that when both tubes were inefficient and both tympanic membranes were compressed inwards there was deafness without vertigo. But when only one Eustachian tube was obstructed, then the deafness was not noticed while the vertigo was prominent, due to the heterogenous stimuli. This is very important to air pilots, submarine and caisson divers. In children with large tonsils and adenoids with obstruction of Eustachian tube, attacks of dizziness can be induced in similar fashion.

c. Tubotympanitis, either by Eustachian tube obstruction or by absorption of toxins through the circulation, oftentimes produces vertigo.

d. In otosclerosis, vertigo is not severe, apparently due to a disturbance in labyrinthine circulation. When severe it is usually due to Eustachian obstruction. When same is opened the vertigo usually subsides.

3. Vertigo associated with inner ear lesions:

a. The noninflammatory diseases of the inner ear caused by toxic factors is not as serious as the inflammatory lesions which produce fatal meningitis.

b. The various forms of inflammatory labyrinthitis are always secondary to a chronic or acute running ear, and the vertigo is always present with the nystagmus. The vertigo is in the same plane and in the same direction of the nystagmus. When the nystagmus is intensified the vertigo is pronounced. When the nystagmus is diminishing the vertigo disappears in the same proportion. Past-pointing and falling in all these cases are in the opposite side of the nystagmus and vertigo. Meningitis and cerebellar abscesses are the common complications.

c. Meniere's symptom complex: Much can be said about this interesting and yet unfortunate syndrome. Many variegated causes, both intracranial and intra-aural, have been advanced. It would be superfluous here to discuss them.

The picture presented by the patient clinically is typical, and the diagnosis is not difficult. Usually occurs in middle age, rarely in youth and old age. During the attack there is deafness, tinnitus aurium, vertigo, nausea, vomiting and no loss of consciousness. This is always unilateral. This attack lasts from several hours to several weeks. It never causes death. During the interim the patient feels fine except complaining of deafness and tinnitus in the involved ear.

Examination will reveal a conductive deafness in the early stage, with a perceptive type deafness in the more chronic cases. The labyrinth on that side will show a loss of function anywhere from 50 per cent to complete destruction.

Today, there are two very important schools, namely: Conservatism versus Radicalism and both report excellent results. It was Charcot in 1874 who recommended resection of the auditory nerve for tinnitus aurium without any success which stimulated Dandy[8] in 1928 to employ this procedure for vertigo. In 42 cases of Meniere's disease resection of the auditory nerve stopped recurrent attacks of vertigo in every case. Some cases ran for years without a single attack. This has been confirmed by other neuro and oto surgeons. Dandy thinks the

pathology is in the vestibular nerve and not in the end organ itself.

Dedering and Mygind, of Denmark, found a water logged condition of the labyrinth due to a faulty water metabolism in these cases of Meniere's syndrome. This would also cause a water logged condition of Eustachian tube with resultant closure increasing intratympanic pressure, which, in turn, increases the intralabyrinthine pressure and in this fashion initiates an attack. Furstenberg[6] with Lashmet and Lathrop from their experiments found that this faulty water metabo-

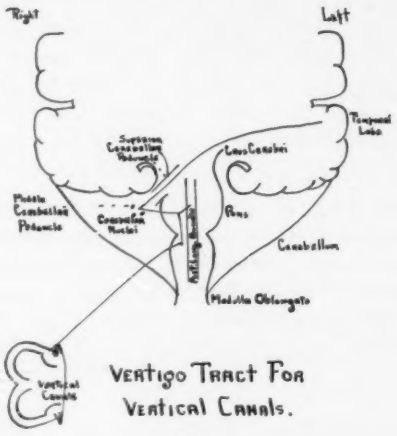

Fig. 3.

lism was due to the retention of sodium by the body. The local tissues involved in Meniere's disease have either an increased avidity for sodium or an unusual sensitivity to it. In 14 typical cases of Meniere's disease, treated by diet, they controlled every attack.

II. Vertigo Associated With C.N.S. Lesions:

Vertigo is a very common symptom of lesions of C.N.S. The vertigo in these conditions is ill defined. A diagnosis is elicited only in conjunction with other neurological findings. Oftentimes a competent neurologist is required for interpretation of these lesions. The most prevalent are:

1. Tumors of Cerebellopontine Angle or VIIIth Nerve Tumor (Symonds[10] and Russell Brain[11]) : Frequency of vertigo varies. In Cushing's series of 33 cases of acoustic tumors the vertigo was present only in five cases. Vertigo is inconstant and a late symptom in these lesions. Vertigo is due more to pressure on the pons than irritation to the vestibular nerve. This is further evidenced by the fact that Dandy in his operation for severance of the nerve handles the same rather firmly and the patients do not complain of vertigo.

The angle syndrome is quite characteristic: *a.* The ear involved fails to elicit any cochlear response. The horizontal and vertical canals fail to produce nystagmus, vertigo, past-pointing and falling. *b.* The sound ear shows normal hearing, stimulation of the left horizontal canal produces normal nystagmus, past-pointing and falling. *c.* Stimulating the vertical canals of the sound ear fails to produce any responses whatever. These fibres lie more superficially than the horizontal fibres, and more easily impaired by tumor pressure on the pons.

2. Lesions of the Pons and Medulla: The most common are: Posterior inferior cerebellar thrombosis, disseminated sclerosis, encephalitis and tumors. The vertigo results from pressure due to congestion and edema. Vertigo is more common in the posterior inferior cerebellar thrombosis than in the others. When the attacks of vertigo occur in these cases they are quite severe. Vertigo *per se* in these cases is of no clinical value whatever.

3. Cerebral Tumors: From frontal, parietal and temporal lobes, vertigo is uncommon, and when present assumes no clinical value.

4. Traumatic Injuries: Vertigo is practically constant in 90 per cent of traumatic injury to the head (Brain,[11] Linthicum[12] and Rand and Grove[4]). It forms one of the most important symptoms in cerebral contusion. It assumes no definite form. Dizziness in the vast majority of these cases arises from trauma along the intracranial ramification of the vestibular nerve, rather than in the peripheral organ. Vertigo originating from a peripheral lesion has a good prognosis, whereas coming from a central involvement has a poor prognosis.

5. Syphilis: The syphilitic toxins have a specific attraction for the C.N.S. without any manifestation of local syndrome.

Tabes or paresis usually result in later years from this type of case. The vestibular nerve is involved very early in the disease.

6. Peripheral Vertigo May Be Differentiated from Central Vertigo: a. Spontaneous vertigo of labyrinthine (peripheral) origin is always associated with spontaneous nystagmus and is always increased when the eyes are turned in the direction of the nystagmus. Spontaneous vertigo when not accompanied by nystagmus or unable to elicit nystagmus by vestibular tests speaks for central involvement (Kerrison[13]).

b. Spontaneous vertigo with spontaneous nystagmus and the labyrinth eliciting normal response speaks for central lesion.

c. Spontaneous vertigo associated with a perverted or inverse nystagmus speaks for a central lesion.

Perverted nystagmus or inverse nystagmus is pathognomonic of a central lesion and is indicative of brain stem involvement. Perverted nystagmus means, for example, douching the horizontal canal and instead of obtaining a normal horizontal nystagmus, a vertical nystagmus is elicited. An inverse nystagmus is present when douching the right horizontal canal with cold water and obtain a horizontal nystagmus on the same side instead of going to the opposite side.

d. The failure to past-point correctly after a normal induced nystagmus associated with spontaneous vertigo speaks for central lesion.

e. The marked derangement of vestibular responses with the presence of a normal or slightly altered cochlear response in the presence of spontaneous vertigo speaks for a central lesion.

f. Spontaneous vertigo, not increased, after induction of marked nystagmus by turning or by a caloric test, and with the absence of the vegetative reflexes, *viz.*, nausea, vomiting, pallor, etc., speaks for central involvement.

g. In labyrinth lesions vertigo is a more dominant symptom than motor disturbances of equilibrium; whereas, in cerebellar lesions the opposite is true (Thomas[6]).

h. In cerebellar abscesses irritative signs are evident over a prolonged period of time; whereas, in acute labyrinthitis the irritation is short.

i. In acute labyrinthitis vertigo follows an orderly course, progresses rapidly, until destruction is complete, remains for a while at a maximum, then gradually diminishes. In cerebellar abscess vertigo, etc., increases as the disease advances, but unevenly; there are remissions and exacerbations.

j. When labyrinth function is lost and vertigo and nystagmus persist or increase, and especially if signs of intracranial hypertension are present, a cerebellar abscess should be suspected.

III. VERTIGO ASSOCIATED WITH OCULAR LESIONS:

1. Any kind of eye muscle imbalance can cause vertigo. *2.* Eye muscle paresis. *3.* Eye muscle paralysis.

The simplest test to differentiate ocular vertigo from aural or central vertigo is to close the eyes, and if the vertigo present disappears then it is ocular. If same remains, it is either aural or central (Mackenzie[14]).

These ocular lesions give rise to a diplopia, which confuses the patient with resultant vertigo. This is not a true vertigo for the aural apparatus or any of its ramifications are not involved.

IV. VERTIGO ASSOCIATED WITH SYSTEMIC LESIONS:

a. Cardiovascular conditions: Dizziness may be a symptom of cardiovascular lesions, but the latter is never the cause of the former. This condition cannot produce vertigo. Vertigo is essentially a condition that arises from the ear, or from its tracts or from its cortical centre. Vertigo in these cases results not from the nature of the disease, but solely from an insufficient blood supply to the labyrinth.

Vertigo is common in arteriosclerosis, especially when there is a sudden great drop. The danger here is not to lower the blood pressure any more by sedation during the attack, but to increase it by stimulation. Here vertigo is due to an anemia of the labyrinth and brain. However, high blood pressure, which causes sudden cerebral hyperemia on slight exertion, may result in vertigo, and in these cases, sedation is indicated instead of stimulation.

b. Digestive causes: Neurovisceral impulses coming from the stomach, etc., passes to the vestibulosympathetic system, to

the phrenic and vagus nerves, thereby producing the vegetative symptoms, *viz.*, pallor, sweating, nausea, vomiting and faintness, etc. This is a reflex vertigo. Conversely, a running ear can easily produce this syndrome with a normal gastric intestinal apparatus, through its irritation of the vestibular nerve.

c. Nose and throat conditions can affect sphenopalatine ganglion, which has connections with tympanic plexus, and thereby may produce vertigo by affecting unilaterally the labyrinth circulation.

d. Toxic factors: *1.* Drugs: Tobacco, quinine, salicylate, atropine, arsenic, carbon monoxide, lead, mercury and other heavy metals. *2.* Bacteria: Teeth, tonsils, sinus, gall bladder, appendix, colon, mumps in acute cases and nephritis. *3.* Metabolic disturbances: Gout and hypoglycemia diseases. *4.* Allergy: Rather rare, but cases have been reported.

SUMMARY.

1. Equilibrium depends upon three senses: *a.* Kinetic static; *b.* ocular; *c.* kinesthetic.

2. Systematized or tactile forms of vertigo, *per se*, are of no clinical value.

3. Pathological etiologic factors producing vertigo are: *a.* Inflammation; *b.* hyperemia; *c.* ischemia; *d.* faulty water metabolism; *e.* pressure; *f.* reflex phenomenon.

4. Vertigo is essentially an ear problem. Ninety per cent of cases due to aural lesions.

5. Clinically vertigo can be classified and diagnosed with: *a.* Vertigo associated with aural lesions; *b.* vertigo associated with C.N.S. lesions; *c.* vertigo associated with ocular lesions; *d.* vertigo associated with systemic lesions.

6. Peripheral vertigo in many cases may be differentiated from central vertigo.

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1021 Park Avenue.

AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

The American Academy of Ophthalmology and Otolaryngology will meet in New York City, Sept. 26 to Oct. 3, 1936. The headquarters will be at the Waldorf Astoria Hotel. Dr. W. P. Wherry, Executive Secretary, Treasurer.

THE NEW YORK ACADEMY OF MEDICINE.

SECTION OF OTO-LARYNGOLOGY.

Meeting of May 15, 1935.

Case Presentations: a. A Brief Summary of End-Results of Cases of Neoplasm of the Nasal Accessory Sinuses, Treated by Surgery and Radiation. Dr. G. Allen Robinson.

(To be published in a subsequent issue of THE LARYNGOSCOPE.*)*

DISCUSSION.

DR. GEORGE A. WYETH: Neoplastic disease of the antrum and other accessory sinuses attacks alike bone and soft parts. It, therefore, always has been a very troublesome condition for both the radiologist and the surgeon. Dr. Robinson in his presentation of these remarkable cases this evening has shown us not only the cases themselves but has reported a five-year cure in 20 per cent of his 69 cases, which is an arresting achievement. I am sure he is to be congratulated very heartily; however, the importance of his report, it seems to me, is understood fully only when we realize that these were rescued or salvaged cases, that is, cases which had had operative interference. Through his ability with radium he has been able to salvage this large proportion of them.

In his pathological exhibit I was impressed with the number of kinds of neoplasms presented, and I want particularly to emphasize the advisability of a pathological study of each case before treatment is instituted. Whether you believe in the pathological grading of malignancy or not, certainly it is true that the clinician is able to differentiate under the microscope a high grade tumor from a low grade tumor. This information is easily obtained, and this information is of untold value to the progressive surgeon. It is a simple matter, now an office procedure, to obtain a small piece of tissue with the biopsy electrode, make a frozen section and examine it under the microscope. One can discern three things, the three points on which you base your treatment before instituting it — the kind of malignancy, the grade of malignancy and whether the tumor is radiosensitive or radioresistant. The surgeon knows that it is always the high grade tumor which recurs early, metastasizes rapidly and kills quickly. He accepts this warning and no longer attempts to remove with the scalpel a high grade tumor which is radiosensitive, for in these cases the scalpel alone seldom succeeds. Here preoperative radiation is always indicated.

We do not know what proportion of Dr. Robinson's cases were radiosensitive and what proportion were radioresistant, a very important fact in compiling statistics. It is important because you would get a good five-year percentage of cures if you treated low grade malignancies, while it is difficult to get such a good percentage in high grade malignancies. There is reason to believe, however, that if the patients who have been salvaged had been given the benefits of electrosurgery in the beginning — and I say this advisedly since it is not my opinion alone but that of Dr. Clark, of Philadelphia, and Dr. New, of the Mayo Clinic, — such a large percentage of recurrences would not have occurred. I again felicitate Dr. Robinson on this wonderful presentation this evening.

I thought I was to say a few words, too, on the presentation of Dr. Babcock. I simply want to bring out one point on that: The apparatus is very ingenious. I think it very clever, but I cannot refrain from voicing my conviction that laryngectomy is seldom necessary. Laryngectomy itself has demonstrated that the overwhelming majority of carcinomas of the larynx are of the low grade

variety and for this reason I cannot, in the beginning, deprive a patient of his voice box when it is a simple procedure to open the larynx anteriorly with the cutting current, fold it back and there under sight and touch to desiccate (not coagulate) the malignant lesion, regardless of how large or small it is, provided it is still intrinsic. This does not apply to extrinsic lesions.

b. Demonstration of a Device for Aid of Laryngectomized Patients. Dr. James W. Babcock.

M. W., male, age 57 years, had the misfortune to require the removal of his larynx for squamous celled carcinoma of his left vocal cord, which had become extrinsic. This was done Dec. 8, 1932, and on Feb. 3, 1933, an extensive dissection of the right side of his neck for recurrence in cervical lymph nodes was necessary.

He has done well since and has learned to speak fairly well; however, two things have annoyed him greatly: the anesthesia of the right side of his neck and mastoid region, and he could not blow his nose.

There seems nothing to do about the anesthesia as the nerves were destroyed in the cervical dissection. He has, however, made an ingenious device permitting him to blow his nose, which he will demonstrate. This consists of a tube, with a tight fitting connection for his tracheotomy tube on one end and a mouthpiece on the other, similar to that of an artificial larynx, except that no reed is present to produce sound. By this means he obviates a disability which may sound trivial to many but which is a very real annoyance.

In addition to being able to blow his nose by using this device, the sense of smell is again permitted to function, and he finds that he can be more active without dyspnea.

DISCUSSION.

Dr. James W. Babcock: I would like to answer Dr. Wyeth in that this patient did have an extrinsic carcinoma, and that it has been the experience of some of us at the Presbyterian Hospital that the use of diathermy against cartilage sometimes results in a lot of destruction of the cartilage. We have one case that for six months after having a growth removed by the electric knife instead of the steel knife, has had some obstruction and is unable to breathe except through a tracheotomy tube, although the growth was removed. I do not like the use of electrocoagulation against cartilage.

I would also like to congratulate Dr. Robinson on the prosthesis of the eye. It was the experience of the French during the World War that prosthesis was better than wearing an artificial one. It looks a lot better than any result of plastic surgery would be likely to do.

Report of Acute Infections of the Middle Ear and Mastoid Process at the Manhattan Eye, Ear and Throat Hospital During 1934; Their Prevalence and Virulence. Dr. John R. Page.

(To be published in a subsequent issue of The Laryngoscope.*)*

Intrauterine and Neonatal Otitis With Report of a Case of Otitic Meningitis. Dr. Frederick A. Hemsath.

Seven cases are reported with clinical histories, autopsy findings and microphotographs of the histological sections of the petrous portions of the temporal bones. These cases illustrate the various types of otitis in stillborn and newborn infants.

Two stillborn fetuses and one infant which lived only six hours show a foreign body exudate in the tympanic cavities due to the presence of the soiid constituents of liquor amnii. Two cases, born after premature rupture of membranes, show purulent exudate in addition to cornified cells from the liquor amnii and bacteria are demonstrated in the sections. Pneumonia is present in

one case, not in the other. The sixth case, with death on the sixth day, is one of meningitis secondary to unilateral purulent otitis media and antritis. Bacterial stains of the tissue show gram-positive lancet-shaped diplococci in the right tympanum, right dura mater about the saccus endolymphaticus and the pia-arachnoid about the brain stem. An early bilateral purulent labyrinthitis is present. The last case is one with death on the twenty-second day of pyemia following erysipelas of five days' duration. The tympanic cavities show an organizing exudate in which streptococci appear in tissue stains as agonal invaders.

DISCUSSION.

Dr. Edmund P. Fowler, Jr. (by invitation): I would like to confess that when I was asked to discuss this paper, I did not even know what Dr. Hemsath meant by "cornified cells." When I spoke to some of the obstetricians about it, they told me that at the Sloane Hospital, Johnson and Myers had done quite a lot of work on neonatal pneumonia in infants. I read their paper and found that there is a very interesting literature on the aspiration of amniotic fluid. "Cornified cells," it seems, are just desquamated cells from the skin of the fetus and are pathognomonic of amniotic fluid. I thought if Dr. Hemsath found them in the middle ears of infants, I might have some in my collection. I first went through my series of 15 to see if I could find any cornified cells in the middle ears. It would not surprise Dr. Hemsath but it did surprise me to find that several of them showed amniotic fluid in the middle ear. I looked up the history of one, a five months' embryo. The mother had come into the hospital with ruptured membranes and one foot of the fetus was presenting. It was not known how long the membranes had been ruptured, but it was thought to be quite some time. The baby was delivered as a breech. The heart beat for 40 minutes and the fetus gasped once during that time. Examination under the microscope showed air in the middle ear cavity as well as cornified cells, all kinds of leukocytes, including polymorphonuclears, and the large phagocytic cells one ordinarily finds in the middle ear, and various epithelial cells. I have never been as lucky as Dr. Hemsath in obtaining beautiful bacterial stains such as those on his material. I congratulate him on them.

At any rate, here is a five months' embryo with middle ear disease, which is interesting. Clinically, this has a certain amount of importance. I do not know how many here know the theories of Wittmaack of pneumatization. Dr. Wittmaack places a great deal of stress on this middle ear disease in infants. He says than an infantile mastoid in adults is always due to insufficient pneumatization in infancy. He says that what happens is that neonatal middle ear disease prevents the proper outpouching from the middle ear. I never realized the prevalence of this early otitis media until Dr. Hemsath brought it to my attention. I think there must be more in Prof. Wittmaack's theories than we thought. Many of us believe that inflammation in later life can produce sclerotic mastoids but the paper which Dr. Hemsath has presented tonight suggests that Wittmaack's ideas of faulty development may be more correct than we have thought.

There is one question I would like to ask Dr. Hemsath about his presentation, and that is, how many bacteria are present in the amniotic fluid of mothers whose membranes have been ruptured for a long time? Does he feel that the number of bacteria he found in the middle ear and the amount of foreign body reaction he found are more than he would get from aspiration alone? In only a few of his cases was there much reaction in the membrane of the middle ear itself. There was some question in my mind in studying his sections whether all of the bacteria and all of the cell content of the fluid might not have been direct aspiration from long continued rupture of the membranes, *i.e.*, infection of the amniotic fluid in the mother and then aspiration of that infected fluid by the baby. I could not be sure that there was enough reaction present to justify a conclusion that the amniotic fluid was producing a real otitis media.

Dr. Frederick A. Hemsath: With regard to Dr. Fowler's question as to how many bacteria are present in the amniotic fluid after long ruptured membranes, bacteria rapidly ascend the vagina and invade the amniotic fluid. The group at Sloane Hospital consider membranes long ruptured after two hours,

so that you see when you reach 12 or 24 or even 48 hours, you have a very long period of ruptured membranes. A number of studies have been made on that. I think Dr. Douglas has reported in the past two years on this from Cornell. It depends on how virulent the organism is. I think the suggestion is very important.

There is another aspect which I did not touch on and that is the fact that the infant may have passed meconium. Therefore, there is also meconium in the amniotic fluid and the bile salts it contains are very irritating.

Streptococcus Hemolyticus Bacteremia With Special Reference to Oto-laryngological Conditions. Dr. Joseph L. Goldman and Dr. Gregory Shwartzman.

This study is concerned with 168 cases of streptococcus hemolyticus bacteremia observed in the various medical and surgical departments of the Mount Sinai Hospital during a period of five and one-half years. This constituted about one-fifth of all proved bacteremias during the stated period. Of these cases, 70 or 42 per cent of the total number were either of otitic or pharyngeal origin.

The route of invasion into the blood stream was used for the grouping of the cases. Each group was further studied in relation to age, seasonal incidence, prognosis, and the clinical picture observed. In analyzing the blood cultures the authors were especially interested in the diagnostic and prognostic significance of the number of organisms cultivated from the blood stream.

The authors attributed the high percentage of positive blood cultures obtained in these cases, especially in fluid media alone, to the large quantity of blood drawn, the variety and enrichment of the media employed, the careful adjustment of pH of the media, the daily subcultures, spreads and prolonged observation.

There were 20 cases in the group of upper respiratory infections and 43 in the group of lateral sinus thrombosis. The mortality rates of these two groups were 34 per cent and 37 per cent respectively, as compared to 54 per cent for the 168 cases.

Most of the upper respiratory infections occurred in the winter and spring months. The cases belonging to this and the lateral sinus thrombosis groups also showed a tendency to occur during the early years of life.

The conspicuous groups illustrating the diagnostic value of the blood cultures were the cases of lateral sinus thrombosis and upper respiratory infections. In the cases of lateral sinus thrombosis, 50 per cent of the preoperative blood cultures showed growth in fluid media only, and growth appeared in but one fluid medium in 21 per cent of these cultures. Preoperative blood cultures were positive in 96.7 per cent of the cases in this group. In the cases of upper respiratory infections, bacterial growth was restricted to fluid media in 61 per cent of the cultures.

Of special interest among the cases of upper respiratory infections was the group which developed long bone metastases. The infection in these cases (10) manifested a predilection for young children, a tendency towards complete recovery (90 per cent), and a small number of organisms in the blood stream in a majority of the cases (fluid media only 60 per cent).

The data embodied in the paper disclosed that in contrast to nonhemolytic streptococci (alpha and gamma), the finding of streptococcus hemolyticus (beta) in the blood stream, even in extremely small numbers, was of important clinical significance for diagnosis, prognosis, and indication for surgical interference.

DISCUSSION.

Dr. Emanuel Libman: Before I make the remarks that I desire to, in discussion of this interesting paper, I would like to briefly describe to you the most widely accepted classification of streptococci. Besides the streptococcus hemolyticus or beta streptococcus, there are two nonhemolytic groups, the alpha

and gamma types. The alpha is called streptococcus viridans. It is incorrect to call all non-hemolytic streptococci, "viridans." The viridans (alpha) type presents a green colony, or a green halo about the colony, or both. There is no real hemolysis (I prefer to use the term "clearing"), but there may be a fine clear zone about the colony. The colonies of the gamma type — streptococcus anhemolyticus — are not green, there is no green halo, and no zone of clearing whatsoever.

To me the most interesting group of cases presented tonight are those of pharyngitis with metastatic bone lesions. As regards the question of bacteremia from metastatic infections, that is not of much consequence in otological work. As a rule, when you have stopped the flow in the jugular vein, you are through. Apart from that consideration, it is important to know that many metastases are not apt to set up a secondary bacteremia. In most cases, if a bacteremia persists after a primary focus is operated upon, it will be found that there is still trouble in that situation. The metastatic infections that are apt to throw bacteria back into the blood are osteomyelitis, meningitis, peritonitis, and perhaps purulent pleurisy and prostatic abscess. It is also important to know that a patient may lose a bacteremia, and still go on to a fatal issue. A striking example was a child suffering from otitic meningitis, with 1000 colonies of streptococcus hemolyticus to the ccm. of blood, who lost the bacteremia, and died a few days later. One must not judge the condition of a patient by a bacteremia alone, unless it be excessive.

As regards the percentage of positive cultures in the cases of sinus thrombosis, the figures given here tonight are higher than I obtained. No one can obtain 100 per cent positive results. If anyone claims to, it means that he has excluded all the cases that were not positive, and were not operated upon. To make this point clearer, I will cite the observations made by Dr. Ottenberg. In one case, for example, he found 24 colonies in the jugular vein on the involved side, seven in the other jugular vein, and none in the arm vein. That is enough to show that the cultures from the arm veins cannot be expected to be uniformly positive.

The authors found a large number of cases in which only the cultures in the fluid media were positive, whereas I found only a few. I think that this means that their fluid media were better than mine, or (rather) that the solid media were not as good. This subject needs investigation because I have always regarded the solid media used nowadays as being much more favorable than those formerly employed.

Invasions of the blood by nonhemolytic streptococci must be carefully evaluated. They are significant in cases of subacute bacterial endocarditis, and when an active focus due to them is accompanied by a general infection. In cases of rheumatic fever, I believe that their presence in the blood represents a secondary invasion. They seem to be entering the blood all the time, and accordingly are found as secondary invaders in the greatest variety of diseases. As Dr. Goldman has indicated, they are, as a rule, to be considered as being of significance, if they are found in the plates, but still more so if found more than once, or if present in large numbers.

I would like finally to draw your attention to a small but important group of cases, in which there is found in the flasks an organism that has the morphology of a pneumococcus, and does not survive transplantation. The clinical picture is as follows: Within a few days after operation upon the mastoid bone, there developed high fever, headache and vomiting, but no evidence of meningitis. They end by crisis after about four to five days. This observation was particularly important in a case that I saw a number of years ago with Dr. Whiting. Both mastoid bones had been operated upon, and the lesion was equally deep on both sides. After a few days, there developed the clinical picture just described. The blood culture was reported positive, and there arose the difficulty of making a decision as to which lateral sinus should be explored. When I inquired as to the organism found, I was informed that the bacteriologist had reported the presence in a flask of an organism that looked like a pneumococcus but was not viable. I advised against an operation at the time. A few days later, a crisis occurred, and the patient rapidly recovered. I have no explanation to give for this remarkable picture.

BOOK REVIEWS.

Chronic Nasal Sinusitis and Its Relation to Mental Disorder. F. A. Pickworth, B.Sc., M.B., B.S., A.I.C. (Exam.), Director, Joint Board of Research for Mental Disease, City and University of Birmingham. An Applied Pathology of Abnormal Conditions of the Nasal Sinuses Found in Mental Hospital Patients. With 83 illustrations, seven in three-color and four in two-color. London: H. K. Lewis & Co., Ltd., 1935. Royal 8 vol., 16s net.

In the preface, the author briefly sets forth the object of this book, to demonstrate: First, the existence, extent and complications of chronic nasal sinusitis occurring in mental hospital patients; second, the mechanisms by which such chronic disease results in those physical changes of the brain tissue which are present in cases showing symptoms of mental disorder.

Stress is laid on the correlation of mental disorders to disturbed vascularity of the brain and emphasizes the pathological and biochemical findings in their possible effect upon cerebral circulation.

The author's long experience on the Board of Research for Mental Diseases eminently qualifies him to discuss a subject of vital importance in the development of laryngology, namely, the relation of chronic sinus inflammations and infections to cerebral pathology.

The illustrations are from original specimens and the clinical data gathered from the author's personal service and observations.

It is an excellent monograph on a comparatively recent development of a specialty within specialties. M. A. G.

Behavior Development in Infants. Evelyn Dewey. A Survey of the Literature on Prenatal and Postnatal Activity 1920-1934. Published for the Josiah Macy, Jr., Foundation. New York: Columbia University Press, 1935. $3.50.

"The present survey of the current literature on growth processes and infant behavior is the result of a suggestion of the Josiah Macy, Jr., Foundation, and was made possible by its special grant for this purpose." The present volume is a survey of the literature on prenatal and postnatal activity as collected from 1920 to 1934 and affords valuable reference to subject-matter activity under consideration and study by all fields interested in this important problem.
M. A. G.

Eponyms of Anatomy. Louis R. Effler, A.M., M.D., F.A.C.S., Author of "Three Minute Medicine," "Analogy and Medicine." A Series of Brief Essays on Semi-Popular Medicine Designed Especially for the Freshman Medical Student Beginning Anatomy. With Bibliography, List of Eponyms and Index. Toledo, Ohio: McManus Troup Company. 382 pp.

This volume presents quite an original grouping of anatomical landmarks easily recognized in the nomenclature under the name of their discoverers, such as The Antrum of Highmore, the Organ of Corti, the Membrane of Shrapnell and the Hellicotrema of Cotugno.

Each eponym is briefly described and linked with a biographical sketch of its discoverer and, in most cases, any data of interest that may have a bearing on its discovery or anatomical value.

The volume indicates much painstaking work and delving into the history of anatomy and of its pioneers.

It is originally designed for medical students in the study of anatomy, but much profitable and interesting information may be gleaned by the advanced student of anatomical science in a careful perusal of its pages. M. A. G.

Manipulative Methods in the Treatment of Functional Disease. Edwin Hopewell-Ash, M.D., London, Bachelor of Surgery, London University; Member of the Royal College of Surgeons; Physician-Neurologist to the City of London Red Cross Hospital, 1916-1918; Cheadle Gold Medalist, St. Mary's Hospital, London W.-1. 92 pp. John Bale, Sons & Danielsson, Ltd., 83-91 Gt. Titchfield Street, 1935. 3/6 net.

Physio-Therapy is becoming an all-embracing subject and the author of this monograph seems to classify his manipulative surgery as a branch under this general head.

There has been such a distortion of scientific values in the development of osteopathy, chiropractic and other similar cults, that, in the mind of this reviewer, too liberal encouragement of such innovations must be carefully considered.

The author writes frankly of his manipulative surgery but to the uninitiated it seems to be but variations and modifications of types of massage. M. A. G.

Diseases of the Mouth and Their Treatment. Hermann Prinz, A.M., D.D.S., M.D. D.Sc., Dr. Med. Dent., Professor of Materia Medica and Therapeutics, the Thomas W. Evans Museum and Dental Institute, School of Dentistry, University of Pennsylvania, Philadelphia, and Sigmund S. Greenbaum, B.S., M.D., Associate Professor of Dermatology and Syphilology in the Graduate School of Medicine of the University of Pennsylvania; Attending Dermatologist, Mt. Sinai and Philadelphia General Hospital. A Textbook for Practitioners and Students of Medicine and Dentistry. Illustrated with 287 engravings and 11 colored plates. 602 pp. with Index. Philadelphia: Lea and Febiger, 1935.

There has always been a field in medicine which cannot be assigned exclusively to either medicine or dental professions. This science, diseases of the mouth, still retains its autonomous and neutral position and continues of absorbing interest to the dentist, the oral surgeon, laryngologist and general practitioner.

This new text book has, as the authors express it, one specific objective, namely, "to gather the enormous amount of scattered information concerning this subject and to evaluate carefully these numerous publications according to their intrinsic merit."

The many borderlines in medical science that are involved in diseases of the mouth are given careful consideration and description.

The volume is thoroughly up to date, excellently illustrated; the subject matter well classified and written by authors whose experience and judgment qualify them to discuss this important field authoritatively. M. A. G.

The Good Fight. Raymond Leslie Goldman. 194 pp. New York: Coward-McCann, Inc., 1935. $1.50.

If experience and careful observation are the outstanding qualifications for the production of a literary effort or the recording of facts and outlook, Raymond Leslie Goldman is truly super-qualified.

Crippled by infantile paralysis at the age of four, deafened at 19 and afflicted with chronic diabetis, he has recorded in "The Good Fight" the determination, courage, optimism and true philosophy in surmounting these tremendous handicaps and analyzes the true meaning of happiness.

To review this precious volume is a difficult task; it must be read to be appreciated, and you must observe, in the virile and delightful style of the author, his philosophy, comparatives, ambitions and his superb desire to make of these almost insurmountable handicaps an object for real living and real thinking.

The force of example, as presented by this "good fight," should be a stimulus and whose ideas of happiness are estimated on a much more complacent basis.

M. A. G.

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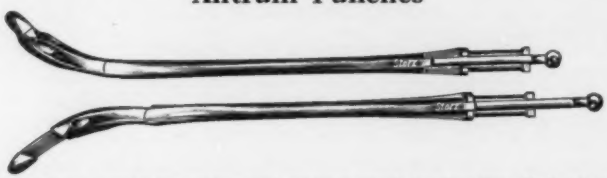

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